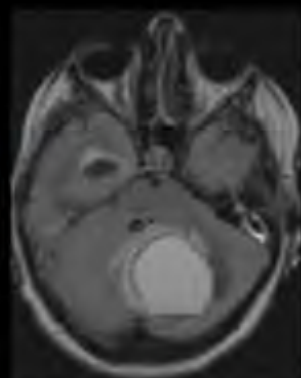


CRACK_{THE} CORE EXAM^{2ND ED}

VOLUMES I & II



STRATEGY GUIDE AND COMPREHENSIVE STUDY MANUAL

PROMETHEUS LIONHART, M.D.

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ALL CHAPTERS WERE WRITTEN BY PROMETHEUS LIONHART, M.D.

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Pediatrics

Prometheus Lionhart, M.D.



Most things in Peds are age dependent. Any time you get a Peds question, the first thing you should do is ask yourself, “what age is the patient?” This is helpful in almost every system; airway infections, liver masses, renal masses, etc..., to help exclude distractors.

Highest yield topics

- Wilms tumor vs Neuroblastoma
- Wilms Associations
- Non-Accidental Trauma
- VACTERL
- Vascular impressions on the trachea / esophagus, barium
- Heterotaxia syndromes

Airway

Croup

This is the most common cause of acute upper airway obstruction in young children. The peak incidence is between 6 months and 3 years (average 1 year). They have a **barky “croupy” cough**. It’s viral. The thing to realize is that the lateral and frontal neck x-ray is done not to diagnosis croup, but to exclude something else. Having said that, the so called “*steeple sign*” - with loss of the normal lateral convexities of the subglottic trachea is your buzzword, and if it’s shown, that will be the finding. Questions are still more likely to center around facts (age and etiology).

Epiglottitis

In contrast to the self limited croup, this one can kill you. It’s mediated by H. Influenza and the classic age is 3.5 years old (there is a recent increase in teenagers - so don’t be fooled by that age). The lateral x-ray will show marked swelling of the epiglottis (*thumb sign*). A fake out is the “omega epiglottitis” which is caused by oblique imaging. You can look for thickening of the aryepiglottic folds to distinguish.

Trivia: Death by asphyxiation is from the aryepiglottic folds (not the epiglottis)

Exudative Tracheitis

This is an uncommon but serious (possibly deadly) situation that is found in slightly older kids. It’s caused by an exudative infection of the trachea (sorta like diphtheria). It’s usually from Staph A. and affects kids between 6-10. The buzzword is *linear soft tissue filling defect within the airway*.

Croup	Epiglottitis	Exudative Tracheitis
6 months - 3 years (peak 1 year)	Classic = 3.5 years, but now seen with teenagers too	6-10 years
Steeple Sign: loss of the normal shoulders (lateral convexities) of the subglottic trachea	Thumb Sign: marked enlargement of epiglottis	Linear soft tissue filling defect (a membrane) seen within the airway
Viral (<i>Most Common - parainfluenza</i>)	H-Flu	Staph. A

Retropharyngeal Cellulitis and Abscess

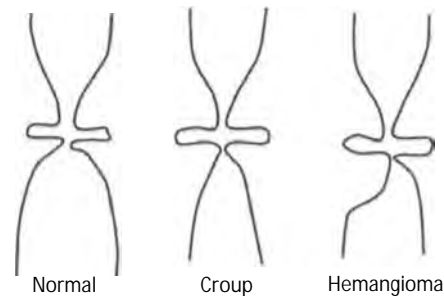
This most commonly affects young kids (age 6 months -12 months). They will most likely show this with a lateral x-ray demonstrating **massive retropharyngeal soft tissue thickening**. For the real world, you can get pseudothickening when the neck is not truly lateral. To tell the difference between positioning and the real thing, a repeat with an extended neck is the next step. On CT it will be very obvious (the fake out would be a more lateral low density suppurative node).



Retropharyngeal Abscess

Subglottic Hemangioma

The hemangioma is the most common soft tissue mass in the trachea, and they are most commonly located in the subglottic region. In croup there is symmetric narrowing with loss of shoulders on both sides (Steeple Sign). In contradistinction, subglottic hemangiomas have loss of just one of the sides.



Trivia

- Tends to favor the left side,
- 50% are associated with cutaneous hemangiomas,
- 7% have the PHACES syndrome.

PHACES

- P- Posterior fossa brain malformation
- H- Hemangiomas
- A- Arterial anomalies
- C- Coarctation of aorta, cardiac defects
- E- Eye abnormalities
- S- Subglottic hemangiomas

Newborn Chest

Meconium Aspiration

This typically occurs secondary to stress (hypoxia), and is *more common in term or post-mature babies (the question stem could say “post term” delivery)*. The pathophysiology is all secondary to chemical aspiration.

Things to know are:

- The buzzword “ropy appearance ” of asymmetric lung densities*
- Hyperinflation with alternative areas of atelectasis*
- Pneumothorax in 20-40% of cases*

Lung Volumes:

High:

- Meconium Aspiration
- Transient Tachypnea
- Neonatal Pneumonia

Low:

- Surfactant Deficiency
- Beta-Hemolytic Pneumonia

Transient Tachypnea of the Newborn (TTN)

The classic clinical scenario is a history of c-section (vagina squeezes the fluid out of lungs normally). Other classic scenario histories include “diabetic mother” and/or “maternal sedation.” Findings are going to start at 6 hours, peak at one day, and be done by 3 days. You are going to see coarse interstitial marking and fluid in the fissures.

Things to know are:

- Classic histories: C-Section, Maternal Sedation, Maternal Diabetes*
- Onset: Peaks at day 1, Resolved by Day 3*
- Lung Volumes - Normal to Increased*

Neonatal Pneumonia (not Beta-Hemolytic Strep)

Lots of causes. Typical look is patchy, asymmetric perihilar densities, and hyperinflation. Will look similar to surfactant deficient disease but will be full term.

Neonatal Pneumonia (Beta-Hemolytic Strep)

This is the most common type of pneumonia in newborns. It's acquired during exit of the dirty birth canal. It affects premature infants more than term infants. It has some different looks when compared to other pneumonias (why I discuss it separately).

Things to know:

- It often has low lung volumes (other pneumonias have high)*
- Granular Opacities is a buzzword (for this and SDD)*
- Often has pleural effusion (SDD will not)*

Surfactant-Deficient Disease (SDD)

This is also called hyaline membrane disease, or RDS. It's a disease of pre-mature kids. The idea is that they are born without surfactant (the stuff that makes your lungs stretchy and keeps alveolar surfaces open). It's serious business and is the most common cause of death in premature newborns. You get **low lung volumes and bilateral granular opacities** (just like B-hemolytic pneumonia). But, unlike B-hemolytic pneumonia you **do NOT get pleural effusions**. As a piece of useful clinical knowledge, a normal plain film at 6 hours excludes SDD.

Surfactant Replacement Therapy

They can spray this crap in the kid's lungs, and it makes a huge difference (decreased death rate etc...). Lung volumes get better, and granular opacities will clear centrally after treatment. The post-treatment look of bleb-like lucencies can mimic PIE.

Things to know:

- *Increased Risk of Pulmonary Hemorrhage*
- *Increased Risk of PDA*

Persistent Pulmonary HTN

Also called, persistent fetal circulation. Normally the high pulmonary pressures seen in utero (that cause blood to shunt around the lungs), decrease as soon as the baby takes his/her first breath. Dr. Goljan (Step 1 wizard) used to call this a "miracle", and used this physiology to deny evolution. When high pressures persist in the lungs it can be primary (the work of Satan), or secondary from hypoxia (meconium aspiration, pneumonia, etc...). The CXR is going to show the cause of the pulmonary HTN (pneumonia), rather than the HTN itself.

Complications of Life in the NICU

Pulmonary Interstitial Emphysema (PIE)

When you have surfactant deficiency and they put you on a ventilator (which pulverizes your lungs with PEEP), you can end up with air escaping the aveoli and ending up in the interstitium and lymphatics. On CXR it looks like **linear lucencies (buzzword)**. It's a warning sign for impending Pneumothorax. Most cases of PIE occur in the first week of time (bronchopulmonary dysplasia - which looks similar - occurs in patients older than 2 weeks). Surfactant therapy can also mimic PIE. The treatment is to switch ventilation methods and or place them affected side down.

A total zebra is the progression of PIE to a large cystic mass. The thing can even cause mediastinal mass effect.

Chronic Lung Disease (Bronchopulmonary Dysplasia - BPD)

This is the kid born premature (with resulting surfactant deficiency), who ended up on a ventilator. Prolonged ventilation in a tiny (<1000 grams), premature kid (<32 weeks) is your classic scenario. So, after week two you start to get hazy lungs, which over the next few months coarsen and give you some bubble like lucencies. **"Band like opacities" is a buzzword.**

Wilson-Mikity Syndrome is a thing that may or may not be real, and/or be changing in the current thinking of what it is. Anyway, historically, it's BPD without being on a ventilator. The CORE is not going to ask you this, just forget I brought it up.

Congenital Chest

Pulmonary Hypoplasia

This can be primary or secondary. Secondary causes seem to lend themselves more readily to multiple choice questions. Secondary causes can be from decreased hemi-thoracic volume, decreased vascular supply, or decreased fluid. The most common is the decreased thoracic volume, typically from a space occupying mass such as a **congenital diaphragmatic hernia** (with bowel in the chest), but sometimes from a neuroblastoma or sequestration. Decreased fluid, refers to the **Potter Sequence** (no kidneys -> no pee -> no fluid -> hypoplastic lungs).

Bronchopulmonary Sequestration

These are grouped into intralobar and extralobar with the distinction being which has a pleural covering. The venous drainage is different (intra to pulmonary veins, extra to systemic veins). **You can NOT tell the difference radiographically.** The *practical difference is age of presentation*; intralobar presents in adolescence or adulthood with recurrent pneumonias, extralobar presents in infancy with respiratory compromise.

- Intralobar:** Much more common (75%). Presents in adolescence or adulthood as recurrent pneumonias (bacteria migrates in from pores of Kohn). **Most commonly in the left lower lobe** posterior segment (2/3s). Uncommon in the upper lobes. In contradistinction from extralobar sequestration, it is rarely associated with other developmental abnormalities.

- Extralobar:** Less common of the two (25%). Presents in infancy with respiratory compromise (primarily because of the associated anomalies - Congenital cystic adenomatoid malformation (CCAM), congenital diaphragmatic hernia, vertebral anomalies, congenital heart disease, pulmonary hypoplasia). It rarely gets infected since it has its own pleural covering. These are sometimes described as part of a bronchopulmonary foregut malformation, and may actually have (rarely) a patent channel to the stomach, or distal esophagus.

Gamesmanship: I say recurrent pneumonia in same area, you say intralobar sequestration.

Bronchogenic Cysts

Typically an incidental finding. They are generally solitary and unilocular. They typically do NOT communicate with the airway, so if they have gas in them you should worry about infection.

Congenital Cystic Adenomatoid Malformation (CCAM)

As the name suggests it's a malformation of adenomatoid stuff that replaces normal lung. Most of the time it only affects one lobe. There is no lobar preference (unlike CLE which favors the left upper lobe). There are cystic and solid types (type 1 cystic, type 3 solid, type 2 in the middle). There is a crop of knuckle heads who want to call these things CPAMs and have 5 types, which I'm sure is evidence based and will really make an impact in the way these things are treated. CCAMs communicate with the airway, and therefore fill with air. Most of these things (like 90%) will spontaneously decrease in size in the third trimester. The treatment (at least in the USA) is to cut these things out, because of the iddy bitty theoretical risk of malignant transformation (pleuropulmonary blastoma, rhabdomyosarcoma).

Congenital Lobar Emphysema (CLE)

The idea behind this one is that you have bronchial pathology (maybe atresia depending on what you read), that leads to a ball-valve anomaly and progressive air trapping. On CXR, it looks like a lucent, hyperexpanded lobe.

Things to know:

- *It's not actually emphysema - just air trapping secondary to bronchial anomaly*
- *It prefers the left upper lobe*
- *Treatment is lobectomy*

Gamesmanship

• *They can show you a series of CXRs. The first one has an opacity in the lung (the affected lung clears fluid slower than normal lung). The next x-ray will show the opacity resolved. The following x-ray will show it getting more and more lucent. Until it's actually pushing the heart over. This is the classic way to show it in case conference, or case books.*

Congenital Diaphragmatic Hernia (CDHs)

Most commonly they are Bochdalek type. B is in the Back - they are typically posterior and to the left. The appearance on CXR is usually pretty obvious.

Things to know:

- *Usually in the Back, and on the left (Bochdalek)*
- *If it's on the right - there is an association with GBS Pneumonia*
- *Mortality Rate is related to the degree of pulmonary Hypoplasia*
- *Most have Congenital Heart Disease*
- *Essentially all are malrotated*

Gamesmanship

- *One trick is to show the NG tube curving into the chest.*

Special Situations in Peds Chests

Viral - In all ages this is way more common than bacterial infection. Peribronchial edema is the buzzword for the CXR finding. "Dirty" or "Busy" Hilum. You also end up with debris and mucus in the airway which causes two things (1) hyperinflation and (2) subsegmental atelectasis.

Round Pneumonia - Kids get round pneumonia. They love to show this, and try to trick you into thinking it's a mass. Younger than 8 you are thinking round pneumonia, round pneumonia, round pneumonia - with S. Pneumonia being the culprit. The PhD trivia is that these occur because you don't have good collateral ventilation pathways. Round pneumonia is usually solitary, and likes the posterior lower lobes. Take home message: *No CT to exclude cancer, just get a follow up x-ray.*

Swyer James - This is the classic unilateral lucent lung. It typically occurs after a viral lung infection in childhood resulting in post infectious obliterative bronchiolitis. The size of the affected lobe is smaller than a normal lobe (it's not hyperexpanded).

Papillomatosis - Perinatal HPV can cause these soft tissue masses within the airway and lungs. It's also seen in adults who smoke. "*Multiple lung nodules which demonstrate cavitation* " is the classic scenario. Some testable trivia includes the 2% risk of squamous cell cancer, and that manipulation can lead to dissemination. The appearance of cysts and nodules can look like LCH (discussed more in the thoracic chapter), although the trachea is also involved.

Cystic Fibrosis- So the sodium pump doesn't work and they end up with thick secretions and poor pulmonary clearance. The real damage is done by recurrent infections.

Things to know:

- Bronchiectasis (begins cylindrical and progresses to varicoid)*
- It has an apical predominance (lower lobes are less affected)*
- Hyperinflation*
- They get Pulmonary Arterial Hypertension*
- Mucus plugging (finger in glove sign)*
- Men are infertile (vas deferens is missing)*

Primary Ciliary Dyskinesia - The motile part of the cilia doesn't work. They can't clear their lungs and get recurrent infections. These guys have lots of bronchiectasis just like CF. BUT, this time its lower lobe predominant (CF was upper lobe).

Things to know:

- Bronchiectasis (lower lobes)*
- 50% will have Kartageners (situs inversus). So, 50% will not*
- Men are infertile (sperm tails don't work)*
- Women are subfertile (cilia needed to push eggs around)*

Sickle Cell / Acute Chest - Kids with sickle cell can get "Acute chest." Acute chest actually occurs more in kids than adults (usually between age 2-4). This is the leading cause of death in sickle cell patients. Some people think the pathology is as such: you infarct a rib -> that hurts a lot, so you don't breath deep -> atelectasis and infection. Others think you get pulmonary microvascular occlusion and infarction. Regardless, if you see opacities in the CXR of a kid with sickle cell, you should think of this.

Gamesmanship (how do you know it's sickle cell?)

- Kid with Big Heart*
- Kid with bone infarcts (look at the humeral heads)*
- Kid with H shaped vertebra (look on lateral)*

Bronchial Foreign Body: The key concept is that it causes **air trapping**. The lung may look more lucent (from air trapping) of the affected side. You **put the affected side down and it will remain lucent** (from air trapping). Another random piece of trivia, is that under fluoro the mediastinum will shift AWAY from the affected side on expiration.

Mediastinal Masses

Anterior.

•**Normal Thymus:** This is the most common mediastinal “mass.” It’s terribly embarrassing to call a normal thymus a mass, but can actually be tricky sometimes. It can be pretty big in kids less than 5 (especially in infants). Triangular shape of the thymus is sometimes called the “sail sign.” Not to be confused with the other 20 sail signs in various parts of the body, or the spinnaker sail sign, which is when pneumomediastinum lifts up the thymus.

•*Things that make you think abnormal*

- Abnormal Size for patients Age (really big in a 15 year old)*
- Heterogenous appearance*
- Calcification*
- Compression of airway or vascular structure*

•**Thymic Rebound:** In times of acute stress (pneumonia, radiation, chemotherapy, burns), the thymus will shrink. In the recovery phase it will rebound back to normal, and sometimes larger than before. During this rebound it **can be PET avid**.

•**Lymphoma:** This is the **most common abnormal mediastinal mass in children** (older children and teenagers). Lymphoma vs Thymus can be tricky. Thymus is more in kids under 10, Lymphoma is seen more in kids over 10. When you get around age 10, you need to look for cervical lymph nodes to make you think lymphoma. If you see calcification, and the lesion has NOT been treated you may be dealing with a teratoma. Calcification is uncommon in an untreated lymphoma.

•*Complications: Compression of SVC, Compression of Pulmonary Veins, Pericardial Effusion, Airway Compression.*

Middle:

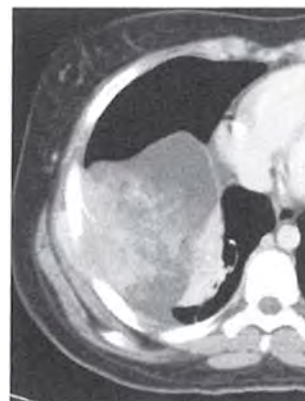
- Lymphadenopathy** - Middle mediastinal lymphadenopathy is most often from granulomatous disease (TB or Fungal), or from lymphoma.
- Duplication Cysts** - These fall into three categories (a) bronchogenic, (b) enteric, (c) neuroenteric. The neuroenterics are posterior mediastinal, the other two are middle mediastinal.
- Bronchogenic** - water attenuation - close to the trachea or bronchus. Bronchogenic cysts tend to be middle mediastinal (esophageal cysts tend to be posterior mediastinal).
- Enteric** - water attenuation close to the esophagus (lower in the mediastinum)

Posterior:

- Neuroblastoma** - This is the most common posterior mediastinal mass in a child under 2. This is discussed in complete detail in the GU PEDs section. I'll just mention that compared to abdominal neuroblastoma, thoracic neuroblastoma has a better outcome. They may involve the ribs and vertebral bodies. Also, remember that Wilms usually mets (more than neuroblastoma) to the lungs, so if it's in the lungs don't forget about Wilms.
- Ewing Sarcoma** - *This is discussed in complete detail in the MSK PEDs section.*

•**Askin Tumor (Primitive Neuroectodermal tumor of the chest wall):** This is now considered part of the Ewing Sarcoma spectrum, and is sometimes called an Ewing sarcoma of the chest wall. They tend to displace adjacent structure rather than invade early on (when they get big they can invade). They look heterogenous, and the solid parts will enhance.

•**Neuroenteric Cyst** - By convention these are associated with vertebral anomalies. The cyst does NOT communicate with CSF, is well demarcated, and is water density.



Askin Tumor

•**Extramedullary Hematopoiesis** - This occurs in patients with myeloproliferative disorders or bone marrow infiltration (including sickle cell). Usually, this manifests as a big liver and big spleen. However, in a minority of cases you can get soft tissue density around the spine (paraspinal masses), which are bilateral, smooth, and sharply delineated

Primary Lung Tumors:

Pleuropulmonary Blastoma - This is a primary intrathoracic malignancy. They can look a lot like CCAMs and even have different types (cystic, mixed, solid). These things are usually right sided, pleural based, and without chest wall invasion or calcifications. No rib invasion, No calcification. The more solid types can have mets to the brain and bones. The cystic type seem to occur more in kids less than a year old, and be more benign.

Trivia:

- 10% of the time they have a multilocular cystic nephroma.

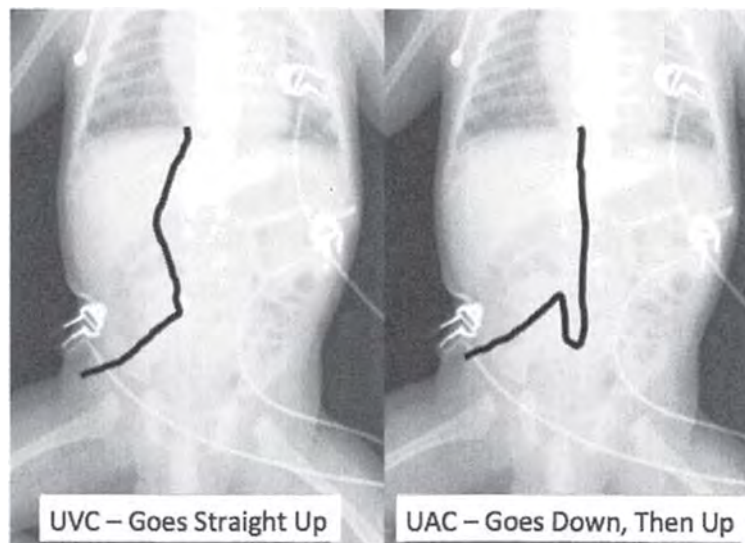
Catheters/Lines

Umbilical Venous Catheter (UVC) - A UVC passes from the umbilical vein to the left portal vein to the ductus venosus to a hepatic vein to the IVC. You don't want the thing to lodge in the portal vein because you can infarct the liver. The ideal spot is at the IVC - Right Atrium junction.

Umbilical Artery Catheter (UAC) - A UAC passes from the umbilicus, down to the umbilical artery, into an iliac artery then to the aorta. Positioning counts, as the major risk factor is renal arterial thrombosis. You want to avoid the renal arteries by going high (T8-T10), or low (L3-L5)

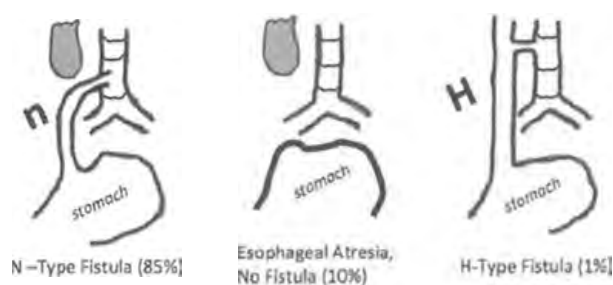
Things to know about UACs:

- It goes down first
- It should be placed either high (T8-T10) or low (L3-L5)



GI

Esophageal Atresia / TE fistula: This can occur in multiple subtypes, with the classic ways of showing it being a **frontal CXR with an NG tube stopped in the upper neck**, or a fluoro study (shown lateral) with a blind ending sac or communication with the tracheal tree.



There are 5 main subtypes, only 3 of which are worth knowing (being familiar with) for the purpose of the exam.

Things to Know About Esophageal Atresia / TE Fistula:

- *Most Important Thing To Know are the VACTERL associations (more on this later)*
- *The most common subtype is the N-Type (blind ended esophagus, with distal esophagus hooked up to trachea)*
- *Excessive Air in the Stomach = H type (can also be with N type)*
- *No Air in the Stomach = Esophageal Atresia*
- *The presence of a right arch (4%) must be described prior to surgery (changes the approach).*

VACTERL: This is extremely high yield. VACTERL is a way of remembering that certain associations are seen more commonly when together (when you see one, look for the others).

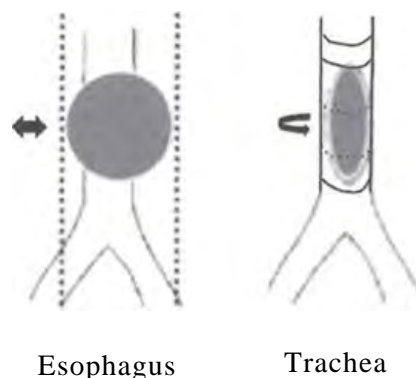
They occur with different frequency:

- V - Vertebral Anomalies (37%)
- A - Anal (imperforate anus) (63%)
- C - Cardiac (77%)
- TE - Tracheoesophageal Fistula or Esophageal Atresia (40%)
- R - Renal (72%)
- L - Limb (radial ray) - 58%

Trivia: If both limbs are involved, then both kidneys tend to be involved. If one limb is involved, then one kidney tends to be involved.

Esophageal Foreign Bodies: Kids love to stick things in their mouths (noses and ears). This can cause a lot of problems including direct compression of the airway, perforation, or even fistula to the trachea. Stuff stuck in the esophagus needs to be removed.

One trick to be aware of is that on a frontal CXR, a coin that is seen totally in the coronal plane is more likely in the esophagus. A coin seen turned 90 degrees is more likely in the trachea (because in the rigid trachea it must turn long ways against the elastic posterior membrane).



Additional trivia relates to swallowed batteries and magnets.

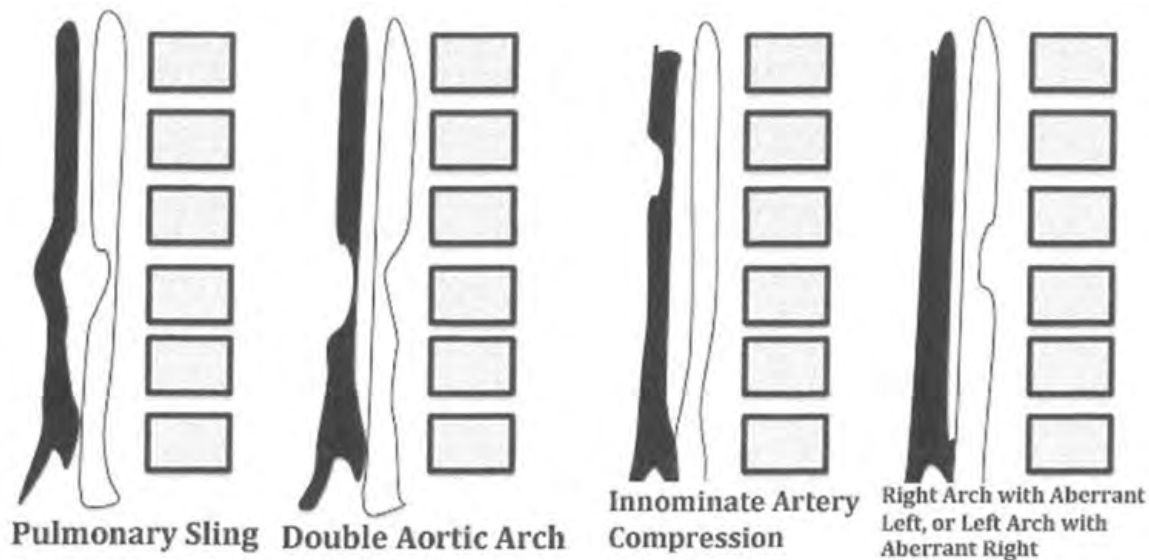
•**Swallowed Magnets** - One magnet is ok. Two or more magnets is a problem. The reason is that they can attract each other across intestinal walls leading to obstruction, necrosis, perforation, and a law suit. They need a surgical consult. Also... MRI is contraindicated.

•**Disc Batteries** - They look like coins, except they have two rings. The literature is not clear, but it appears that modern batteries rarely leak (leaking is bad - caustic chemicals, heavy metals etc.). So most people will watch the transit with serial x-rays. If it gets stuck, they go and get it. If you leave it in longer than a week or so, the risk of it leaking increases.



Disc Battery Coin

Vascular Impression: This is a very high yield topic for the purpose of multiple choice exams. It's important to stress that the **pulmonary sling is the only variant that goes between the esophagus and the trachea**. A classic question, is that this is **associated with trachea stenosis**.



Bowel Obstruction (in the neonate)

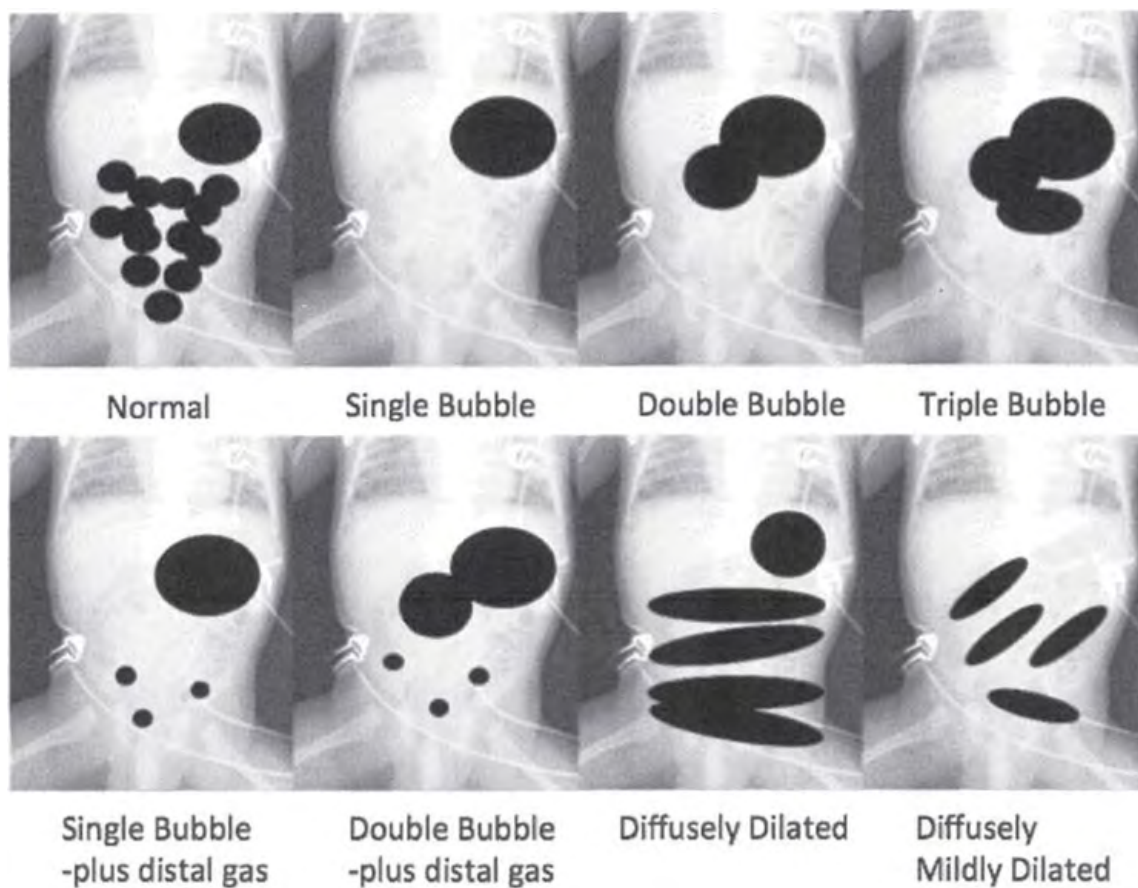
Bowel obstruction in the neonate can be thought of as either high or low. The following sections will walk through an algorithm, starting with plain films for diagnosis (and sometimes management).

Neonatal Obstruction	
High	Low
Midgut Volvulus / Malrotation	Hirschsprung Disease
Duodenal Atresia	Meconium Plug Syndrome
Duodenal Web	Ileal Atresia
Annular Pancreas	Meconium Ileus
Jejunal Atresia	Anal Atresia / Colonic Atresia

The Bubble Trouble of Peds Radiology

People who do peds radiology are obsessed with bubbles on baby grams. The idea is to develop a pattern based approach to bowel obstruction in the newborn.

At the institution I trained at, this was taught as 8 classic patterns:



Single Bubble = Gastric (antral or pyloric) atresia.

Double Bubble = Duodenal Atresia (highly specific). Some authors will say that UGI is not necessary because of how highly specific this is. The degree of distention will be more pronounced than with midgut volvulus (which is a more acute process). Thought to be secondary to failure to canalize during development (often an isolated atresia)

Trivia Regarding Duodenal Atresia:

- 30% have Downs
- 40% have polyhydramnios and are premature
- Tiny amount of air in the distal small bowel does NOT exclude the diagnosis.
- On multiple choice test the “double bubble” can be shown on 3rd trimester OB ultrasound, plain film, or on MRI.

Triple Bubble = Jejunal Atresia. When you call jejunal atresia, you often prompt search for additional atresias (colonic). Just remember that jejunal atresia is secondary to a vascular insult during development.

Single Bubble with Distal Gas = Can mean nothing (lotta air swallowing). If the clinical history is bilious vomiting, this is ominous and can be midgut volvulus (surgical emergency). Next test would be emergent Upper GI.

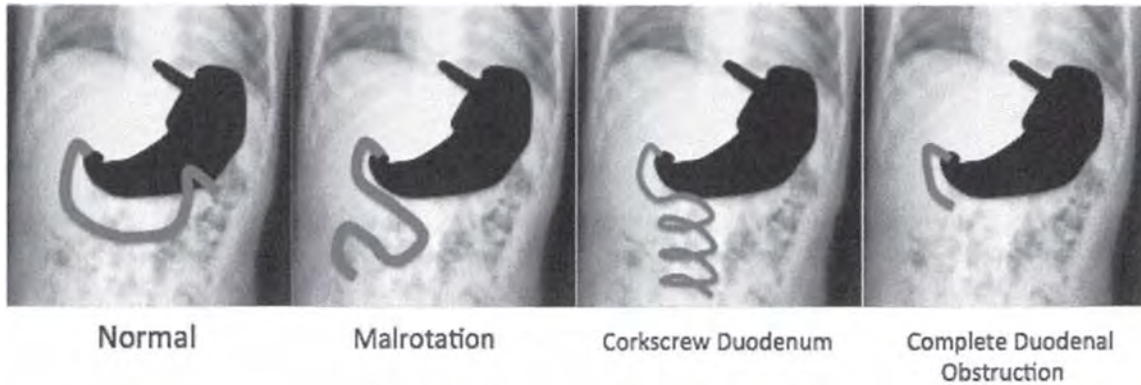
Double Bubble with Distal Gas = Seeing distal gas excludes duodenal atresia. The DDx is a duodenal web, duodenal stenosis, or midgut volvulus. Next step would be upper GI.

Multiple Diffusely Dilated Loops = Suggestive of a low obstruction (ileum or colon). Next step is contrast enema. If the contrast enema is normal you need to follow with upper GI (to exclude an atypical look for midgut volvulus).

Dilated, Scattered Loops = “Sick Belly” - Can be seen with proximal or distal obstruction. Will need Upper GI and contrast enema.

Upper GI Patterns

Upper GI on kids is fair game in multiple choice tests, and real life. Often the answer of this test can equal a trip to the OR for kids, so it's no trivial endeavor.



Malrotation - Normally, the developmental rotation of the gut places the ligament of Trietz to the left of the spine (at the level of the duodenal bulb). These patients are at increased risk for mid gut volvulus, and internal hernias. If you see the appearance of malrotation and the clinical history is bilious vomiting, then you must suspect midgut volvulus.

Trivia regarding Malrotation

- Associated with *Heterotaxy Syndromes*. Associated with *Omphaloceles*.
- Classically shown as the SMA to the right of the SMV (on US or CT).
- False Positive on UGI - Distal Bowel Obstruction, displacing the duodenum (because of ligamentous laxity).

Gamesmanship: *In an infant -*

I say "Non-Bilious Vomiting" — You Say Hypertrophic Pyloric Stenosis

Next Step ? - Ultrasound

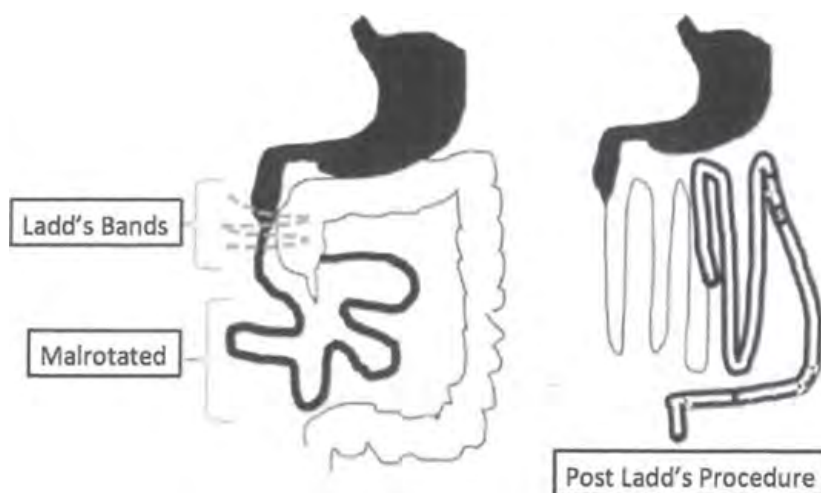
I say "Bilious Vomiting" — You Say Mid Gut Volvulus (till proven otherwise)

Next Step ? - Upper GI

Ladd's Bands - In older children (or even adults) obstruction in the setting of malrotation will present as intermittent episodes of spontaneous duodenal obstruction. The cause is not midgut volvulus (a surgical emergency) but rather kinking from Ladd's Bands.

So what the hell is a "Ladd's Band" ? We are talking about a fibrous stalk of peritoneal tissues that fixes the cecum to the abdominal wall, and can obstruct the duodenum.

Ladd's Procedure - Procedure to prevent mid gut volvulus. Traditionally, the Ladd's Bands are divided, and the appendix is taken out. The small bowel ends up on the right, and the large bowel ends up on the left. They are fixed in place by adhesions (just by opening the abdomen). It is still possible to develop volvulus post Ladd's (but it's rare - 2-5%).

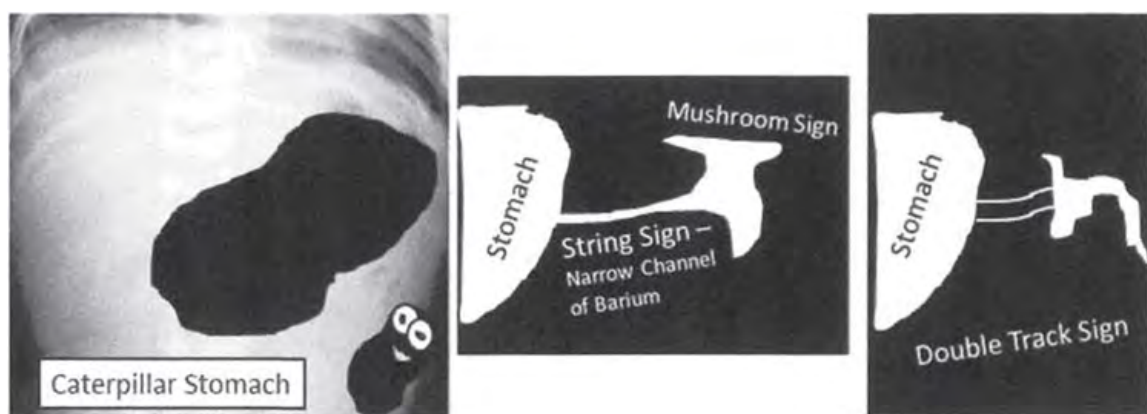


Corkscrew Duodenum - This is diagnostic of midgut volvulus (surgical emergency). The appearance is an Aunt Minnie.

Complete Duodenal Obstruction - Strongly associated with midgut volvulus. If you were thinking duodenal atresia, look for distal air (any will do) to exclude that thought. Plus, as discussed above, you want to see a dilated duodenum (double bubble) for duodenal atresia.

Partial Duodenal Obstruction - If the kid is vomiting this might be from extrinsic narrowing (ladd band, annular pancreas), or intrinsic (duodenal web, duodenal stenosis). You can't tell.

Hypertrophic Pyloric Stenosis - Thickening of the gastric pyloric musculature, which results in progressive obstruction. Step 1 buzzword is “non-bilious vomiting.” Here is the most likely multiple choice trick; this does NOT occur at birth or after 3 months. There is a specific age range 2-12 weeks (peak at 3-6 weeks). Criteria is 4mm and 14mm (4mm single wall, 14mm length). The primary differential is pylorospasm (which will relax during exam). The most common pitfall during the exam is gastric over distention, which can lead to displacement of the antrum and pylorus - leading to false negative. False positive can result from off axis measurement. The phenomenon of “paradoxical aciduria” has been described, and is a common buzzword.

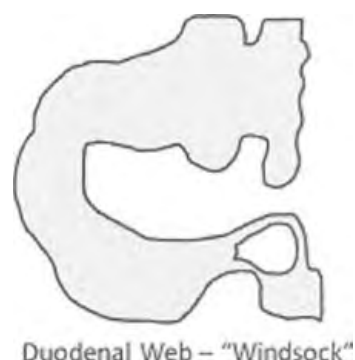


Signs of Hypertrophic Pyloric Stenosis

Gastric Volvulus- This comes in two flavors; organoaxial and mesenteroaxial.

- Organoaxial* - the greater curvature flips over the lesser curvature (rotation along the long axis). This is seen in old ladies with paraesophageal hernias.
- Mesenteroaxial* - twisting over the mesentery (rotation along short axis). Can cause ischemia and needs to be fixed. *This type is more common in kids.*

Duodenal Web: This is a variant of duodenal stenosis, caused by an obstructive duodenal membrane. A pressure gradient causes the formation of the “Wind Sock” (Buzzword).



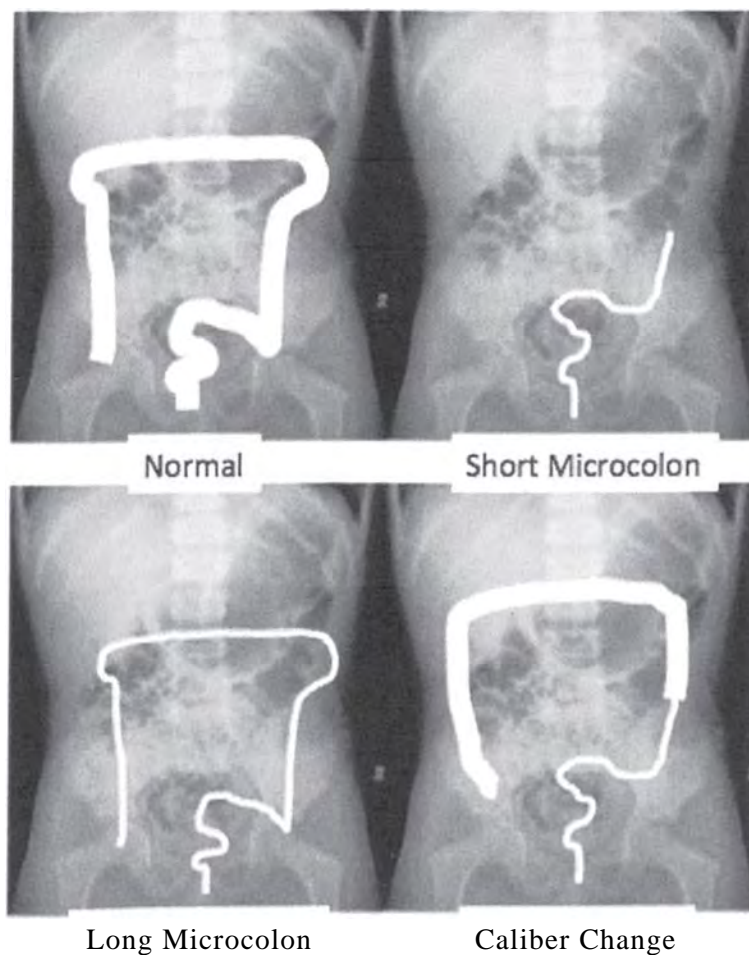
Annular Pancreas: Essentially an embryologic screw up (failure of ventral bud to rotate with the duodenum), that results in encasement of the duodenum. This is a cause of duodenal obstruction in children and pancreatitis in adults. Can also be associated with other vague symptoms (post-prandial fullness, “symptoms of peptic ulcer disease”, etc...). Can be Complete or Incomplete: When it’s incomplete the term “crocodile jaw appearance” is a buzzword.

Can be extramural or intramural:

- Extramural has pancreatic tissue surrounding the duodenum (drains into the main duct).
- Intramural has pancreatic tissue in the wall of the duodenum (drains via small ducts into the lumen).

On imaging, look for an annular duct encircling the descending duodenum.

“Low Obstructions” in a Neonate



Short Microcolon - Think about Colonic atresia

Long Microcolon - This can be seen with meconium ileus or distal ileal atresia.

- Meconium Ileus** - ONLY in patients with CF. The pathology is the result of tenacious meconium causing obstruction of the distal ileum. Contrast will reach ileal loops, and demonstrate multiple filling defects (meconium). This can be addressed with an enema.

- Distal Ileal Atresia** - This is the result of intrauterine vascular insult. Contrast will NOT reach ileal loops. This needs surgery.

Caliber Change - This can be seen with small left colon syndrome or Hirschsprungs

- Small Left Colon Syndrome** - This is a transient functional colonic obstruction, that is self limited and relieved by contrast enema. It is NOT associated with CF. It is seen in infants of diabetic mothers or mothers who have received magnesium sulfate for eclampsia.

- Hirschsprung Disease** - Failure of the ganglion cells to migrate and innervate the distal colon. Affected portions of the colon are small in caliber, with dilation of the normally innervated colon. It's 4:1 more common in boys. There is a 5% association with downs. The key buzzword is rectum smaller than sigmoid or recto-sigmoid ratio < 1 . Another buzzword is "saw tooth" appearance. There is a rare variant, in which the entire colon is involved and can mimic the long microcolon. Diagnosis is made by rectal biopsy. Presentations come in two main flavors: (1) newborn who fails to have BM, (2) One month old who is really sick - with NEC bowel



- Total Colonic Aganglionosis** - This is super rare, and can present with a microcolon. The piece of commonly asked trivia is that it can also affect the terminal ileus.

Meconium Peritonitis: This is a somewhat random GI topic, with a very characteristic look. It's a **calcified mass in the mid abdomen**, traditionally shown on plain film. It is the result of a sterile peritoneal reaction to an intra utero bowel perforation. The bowel perforation could be the result of atresia or meconium ileus. Usually, the perforation seals off prior to birth and there is no leak.

Imperforate or Ectopic Anus: This can range from simple membranous anal atresia to an arrest of the colon as it descends through puborectalis sling. The thing to know is fistula to genitourinary tract. Think VACTERL. Imperforate anus is also associated with a tethered cord (probably need a screening ultrasound).

Obstruction in an Older Child:

Appendicitis - In children older than 4 this is the most common cause for bowel obstruction. If they show this in the PEDs section it's most likely to be on ultrasound. In that case you can expect a blind ending tube, non compressible, and bigger than 6mm.

The classic DDx in "AIM" -

- Appendicitis, Adhesions
- Inguinal Hernia, Intussusception
- Midgut Volvulus, Meckels Diverticulum

Intussusception - The age range is 3 months - 3 years, before or after that you should think of lead points (90% between 3 months and 3 years don't have lead points). The normal mechanism is forward peristalsis resulting in invagination of proximal bowel (the intussusceplum) into lumen of the distal bowel (the intussusciens). They have to be bigger than 3.5cm to matter (in most cases- these are enterocolic), those that are less than 3.5 cm in length are usually small bowel-small bowel and may reduce spontaneously within minutes. Just like an appendix, in the peds section I would anticipate this shown on ultrasound as either the target sign, or pseudo-kidney. Other trivia to think about it as it relates to lead points and reduction. Lead points would be stuff like HSP (vasculitis), meckle diverticulum, enteric duplication cysts.

Reducing Intussusception

- Contraindications: Free Air (check plain film). Peritonitis (based on exam)*
- Recurrence: Usually within 72 hours*
- Success Rates - 80-90% with air*
- Risk of Perforation -0.5%*
- Air causes less peritonitis (spillage of fecal material) than barium*
- Pressure should NOT exceed 120mmHg*

Inguinal Hernia: This is covered in more depth in the GI chapter. Big points are that indirect hernias are more common in kids, they are lateral to the inferior epigastric, and incarceration is the most common complication. Umbilical hernias are common in kids, but rarely incarcerate.

Meckels Diverticulum: This is a congenital true diverticulum of the distal ileum. A piece of trivia is that it is a persistent piece of the omphalomesenteric duct. Step 1 style, “rule of 2s” occurs in 2% of the population, has 2 types of heterotopic mucosa (gastric and pancreatic), located 2 feet from the IC valve, it’s usually 2 inches long (and 2cm in diameter), and usually has symptoms before the child is 2. If it has gastric mucosa (the ones that bleed typically do) it will take up Tc-Pertechnetate just like the stomach (hence the Meckel’s scan).

High Yield Trivia (Regarding Complications)

- *Can get diverticulitis in the Meckels (mimic appendix)*
- *GI Bleed from Gastric Mucosa (causes 30% of symptomatic cases)*
- *Can be a lead point for intussusception (seen with inverted diverticulum)*
- *Can Cause Obstruction*

Special Topics:

Enteric Duplication Cysts - These are developmental anomalies (failure to canalize). They don’t have to communicate with the GI lumen but can. They are most commonly in the ileal region (40%). They have been known to cause in utero bowel obstruction / perforation. Trivia to know - 30% of the time they are associated with vertebral anomalies. If they show it on ultrasound you should think; cyst in abdomen with gut signature. A cyst in the abdomen without gut signature is likely an omental cyst.

Distal Intestinal Obstruction Syndrome - This is one of the many things that can go wrong when you are born with CF. This is sometimes called the “meconium ileus equivalent,” because you end up with a distal obstruction (as the name implies) secondary to dried up thick stool. It more commonly affects the right colon. Kids who get this, are the ones who aren’t compliant with their pancreatic enzymes.

Necrotising Enterocolitis (NEC)- This is a cause of significant mortality and morbidity among small people. It’s thought of as a combination of ischemic and infective pathology, with some people blaming translocation of intestinal bugs through immature mucosa.

Who gets it?

- *Premature Kids (90% within the first 10 days of life)*
- *Low Birth Weight Kids (< 1500 grams)*
- *Cardiac Patients (sometimes occult) - they can be full term*
- *Kids who had perinatal asphyxia*
- *Hirschsprung Kids that go home and come back - they present around month 1.*

What does it look like?

- *Pneumatosis - most definitive finding; Look for Portal Venous Gas Next*
- *Focal Dilated Bowel (especially in the right lower quadrant) - the terminal ileum / right colon is the region most affected by NEC*
- *Featureless small bowel, with separation (suggesting edema).*
- *Unchanging bowel gas pattern - this would be a dirty trick - showing several plain films from progressing days, with the bowel gas pattern remaining the same.*

Useless Trivia:

- *Use of maternal breast milk is the only parameter associated with decreased incidence of NEC.*

Gastroschisis - Refers to extraabdominal evisceration of neonatal bowel (sometimes stomach and liver) through a paraumbilical wall defect.

Trivia to know:

- *It does NOT have a surrounding membrane (omphalocele does)*
- *It's always on the RIGHT side.*
- *Associated anomalies are rare (unlike omphalocele).*
- *Maternal Serum A FP will be elevated (higher than that of omphalocele)*
- *Outcome is usually good*
- *For some reason they get bad reflux after repair.*

Omphalocele - This is a congenital midline defect, with herniation of gut at the base of the umbilical cord.

Trivia to know:

- *It DOES have a surrounding membrane (gastroschisis does not)*
- *Associated anomalies are common (unlike gastroschisis)*
- *Trisomy 18 is the most common associated chromosomal anomaly*
- *Other associations: Cardiac (50%), Other GI, CNS, GU, Turners, Klinefelters, Beckwith- Wiedemann.*
- *Outcomes are not that good, because of associated syndromes.*
- *Umbilical Cord Cysts (Allantoic Cysts) are associated.*

Physiologic Gut Herniation - This is a normal phenomenon that occurs around 6-8 weeks, and can be seen up to 11 weeks. The idea is that the bowel grows faster than the abdominal cavity, and to accommodate this growth it herniates out of the abdominal cavity into the base of the cord. The midgut then rotates and everything comes back to normal.

Trivia to know

- *It's normal*
- *Should NOT contain herniated liver*
- *Should only be seen in early pregnancy (6-8 weeks)*

Mesenteric Adenitis - Self-limiting, usually viral inflammatory condition of mesenteric lymph nodes. It is a classic mimic of appendicitis. The finding is a cluster of large right lower quadrant lymph nodes.

GI Organs

Spleen

Polysplenia and Asplenia - Heterotaxia syndromes are clutch for multiple choice tests. This is discussed in the GI section, but because it's such a high yield topic I'll repeat myself. The major game played on written tests is "left side vs right side." So what the hell does that mean? I like to start in the lungs. The right side has two fissures (major and minor). The left side has just one fissure. So if I show you a CXR with two fissures on each side, (a left sided minor fissure), then the patient has two right sides. Thus the term "bilateral right sidedness." What else is a right sided structure? The liver. So, these patients won't have a spleen (the spleen is a left sided structure). The opposite is true, bilateral left sided patients have polysplenia.

Heterotaxia Syndromes	
Right Sided	Left Sided
Two Fissures in Left Lung	One Fissure in Right Lung
Asplenia	Polysplenia
Increased Cardiac Malformations	Less Cardiac Malformations
Reversed Aorta/IVC	Azygous Continuation of the IVC

Infarcted Spleen - Just say sickle cell.

Liver

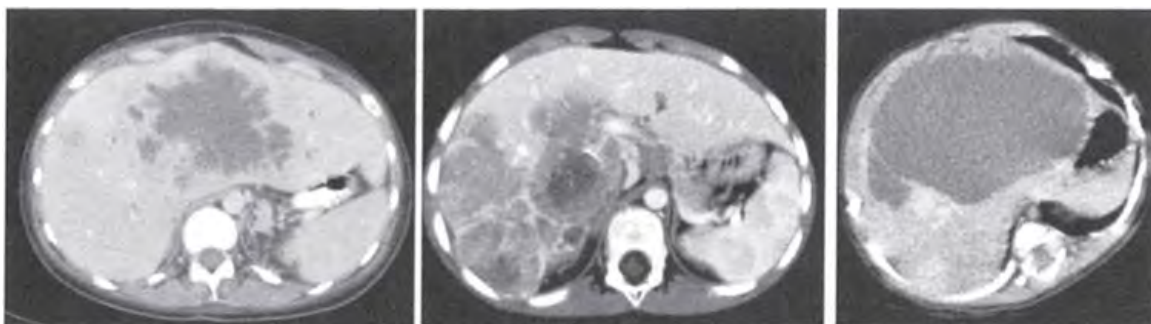
Tumors: With regard to the liver tumors I like to use an age-based system to figure it out. Mass in the liver, first think - what is the age. Then use the narrow DDx to figure it out.

Age 0-3: With kids that are newborns you should think about 3 tumors:

Hemangioendothelioma: Often < 1. Associated with high output CHF, this is classically shown as a large heart on CXR plus a mass in the liver. The Aorta above the hepatic branches of the celiac is often enlarged relative to the aorta below the celiac because of differential flow. They tend to spontaneously involute without therapy over months-years - as they progressively calcify. Skin hemangiomas present 50%. **Endothelial growth factor is elevated.** These can be associated with Kasabach-Merritt Syndrome (the platelet eater).

Hepatoblastoma: Most common primary liver tumor of childhood (< 5). The big thing to know is that it's associated with a bunch of syndromes - mainly hemi-hypertrophy, Wilms, Beckwith-Weidemann crowd. *Prematurity is a risk factor.* This is usually a well circumscribed solitary mass (it can be multiple), that may extend into the portal veins, hepatic veins, and IVC. Calcifications are present 50% of the time. **AFP is elevated.** Another piece of trivia is the hepatoblastoma may cause a precocious puberty from making bHCG.

Mesenchymal Hamartoma: This is the predominately cystic mass (or multiple cysts), sometimes called a "developmental anomaly." Because it's a "developmental anomaly" it shouldn't surprise you that the AFP is negative. Calcifications are UNCOMMON. What is common is a large portal vein branch feeding the tumor.



Hemangioendothelioma

Hepatoblastoma

Mesenchymal
Hamartoma

Age >5

HCC: This is actually the second most common liver cancer in kids. You'll see them in kids with cirrhosis (biliary atresia, fanconi syndrome, glycogen storage disease). AFP will be elevated.

Fibrolamellar Subtype: This is typically seen in a younger patients (<35) without cirrhosis and a normal AFP. The **buzzword is central scar**. The scar is similar to the one seen in FNH with a few differences. This scar does NOT enhance, and is T2 Bright. As a point of trivia, this tumor is Gallium avid. This tumor calcifies more often than conventional HCC.

Undifferentiated Embryonal Sarcoma: This is the pissed off cousin of the mesenchymal hamartoma. It's also cystic, but the mass is much more aggressive. It will be a hypodense mass with septations and fibrous pseudocapsule. This mass has been known to rupture.

Any Age:

Mets: Think about Wilms tumor or Neuroblastoma

Now, there are several other entities that can occur in the liver of young children / teenagers including; Hepatic Adenoma, Hemangiomas, Focal Nodular Hyperplasia, and Angio Sarcoma. The bulk of these are discussed in greater detail in the adult GI chapter.

Congenital:

Choledochal cysts are congenital dilations of the bile ducts -classified into 5 types by some dude named Todani. The high yield trivia is type 1 is focal dilation of the CBD and is by far the most common. Type 2 and 3 are super rare. Type 2 is basically a diverticulum of the bile duct. Type 3 is a "choledochoceles." Type 4 is both intra and extra hepatic. Type 5 is Caroli's, and is intrahepatic only.

Caroli's is an AR disease associated with polycystic kidney disease and medullary sponge kidney. The hallmark is intrahepatic duct dilation, that is large and secular. Buzzword is "**central dot sign**" which corresponds to the portal vein surrounded by dilated bile ducts.

AR Polycystic Kidney Disease: This will be discussed in greater detail in the renal section, but kids with AR polycystic kidney disease will also have cysts in the kidneys, and variable degrees of fibrosis in the liver. The degree of fibrosis is actually the opposite of cystic formation in the kidneys (bad kidneys ok liver, ok kidneys bad liver).



Hereditary Hemorrhagic Telangiectasia (Osier-Weber-Rendu): Autosomal dominant disorder characterized by multiple AVMs in the liver and lungs. It leads to cirrhosis, and a massively dilated hepatic artery. The *lung AVMs set you up for brain abscess*.

Biliary Atresia: If you have prolonged newborn jaundice (> 2 weeks) you should think about two things (1) neonatal hepatitis, and (2) Biliary Atresia. It's critical to get this diagnosis right because they need corrective surgery (Kasai Procedure) prior to 3 months. Patients with biliary atresia really only have atresia of the ducts outside the liver (absence of extrahepatic ducts), in fact they have proliferation of the intrahepatic ducts. They will develop cirrhosis without treatment and not do well.

Trivia to Know about Biliary Atresia:

- Associations with Polysplenia, and Trisomy 18
- Gallbladder may be absent (normal gallbladder — supports neonatal hepatitis)
- Triangle Cord Sign - triangular echogenic structure by the portal vein - possibly remnant of the CBD.
- Hepatobiliary Scintigraphy with 99m Tc-IDA is the test of choice to distinguish (discussed in the Nukes Chapter).
- Alagille Syndrome: This is a total zebra. All you need to know is hereditary cholestasis, from paucity of intrahepatic bile ducts, and peripheral pulmonary stenosis. The purpose of a liver biopsy in biliary atresia is to exclude this diagnosis.
- Gallstones: If you see a pediatric patient with gallstones you should immediately think sickle cell.

If You Remember 4 Things for Peds GI

<i>If You Remember 4 Things for Peds GI</i>	
Absent Gallbladder =	Biliary Atresia
SMA / SMV Reversal =	Malrotation
Absent Spleen / Poly Spleen =	Heterotaxias
VACTERL	

Pancreas

CF - The pancreas is affected in 85-90% of CF patients. Inspissated secretions cause proximal duct obstruction leading to the two main changes in CF: (1) Fibrosis (decreased T1 and T2 signal) and the more common one (2) fatty replacement (increased T1). Patient's with CF diagnosed as adults tend to have more pancreas problems than those diagnosed as children. Those with residual pancreatic exocrine function can have bouts of recurrent acute pancreatitis. Small (1-3mm) pancreatic cysts are common.

High Yield Trivia:

- *Complete fatty replacement is the most common imaging finding in adult CF*
- *Markedly enlarged with fatty replacement has been termed lipomatous pseudohypertrophy of the pancreas. *This is a buzzword.*
- *Fibrosing Colonopathy: Wall thickening of the proximal colon as a complication of enzyme replacement therapy.*



Shwachman-Diamond Syndrome - The 2nd most common cause of pancreatic insufficiency in kids (CF #1). Basically, it's a kid with diarrhea, short stature (metaphyseal chondroplasia pancreas), and eczema. Will also cause lipomatous pseudohypertrophy of the pancreas.

Pancreatitis - The most common cause of pancreatitis in the pediatric setting is trauma (seat belt).

Congenital - Kidneys

Renal Agenesis - Congenital absence of one or both kidneys. If it's unilateral this can be asymptomatic. If it's bilateral think about the "Potter Sequence." When it's unilateral (it's usually sporadic), but for the purpose of multiple choice think about associated GYN anomalies in women (70% of women with unilateral renal agenesis have associated genital anomalies). With regards to men, 20% with renal agenesis have absence of the ipsilateral epididymis and vas deferens or an ipsilateral seminal vesicle cyst.

Associated:

- *Ipsilateral seminal vesicle cysts,*
- *Absent ipsilateral ureter,*
- *Absent ipsilateral hemitrigone*
- *Absent ipsilateral vas deferens*

Potter Sequence: Insult (maybe ACE inhibitors) = kidneys don't form, if kidneys don't form you can't make piss, if you can't make piss you can't develop lungs (pulmonary hypoplasia).

Mayer-Rokitansky-Kuster-Hauser. - Mullerian duct anomalies including absence or atresia of the uterus / unicornuate uterus. Associated with unilateral renal agenesis.

Lying Down Adrenal or “Pancake Adrenal” Sign - describes the elongated appearance of the adrenal not normally molded by the adjacent kidney. It can be used to differentiate surgical absent vs congenital absent.

Horseshoe Kidney - This is the most common fusion anomaly. The kidney gets hung up on the IMA. Complications include Traumatic Injury (gets crushed against vertebral body), UPJ Obstruction, Recurrent Infection, Recurrent Stones, Wilms Tumor (8x higher), TCC (from all those infections). A rare situation, but known association is the renal carcinoid occurring in horseshoe kidney. Turners syndrome is a classically tested association.

Crossed Fused Renal Ectopia - One kidney comes across the midline and fuses with the other. “The Ectopic Kidney is Inferior.” The left kidney more commonly crosses over to the right. Complications include stones, infection, and hyponephrosis (50%).

Prune Belly (Eagle Barrett Syndrome) - This malformation complex occurs in males and includes the following triad:

- Crappy Abdominal Musculature
- Hydronephrosis
- Cryptorchidism (bladder distention interferes with descent of testes)

Congenital UPJ Obstruction - This is the most common congenital anomaly of the GU tract in neonates. About 20% of the time, these are bilateral. Most (80%) of these are thought to be caused by intrinsic defects in the circular muscle bundle of the renal pelvis. Treatment is a pyeloplasty. A Radiologist can actually add value by looking for vessels crossing the UPJ prior to pyeloplasty, as this changes the management.

1970 called and they want to know how to tell the difference between a prominent extrarenal pelvis vs a congenital UPJ obstruction. The Answer is a “Whitaker Test”, which is a urodynamics study combined with an antegrade pyelogram.

Autosomal Recessive Polycystic Kidney Disease (ARPKD) - These guys get HTN, and renal failure. The liver involvement is different than the adult form. Instead of cysts they have abnormal bile ducts and fibrosis. This congenital hepatic fibrosis is ALWAYS present in ARPKD. The **ratio of liver and kidney disease is inverse**. The worse the liver is the better the kidneys do. The better the liver is the worse the kidneys are. Death is often from portal hypertension. On ultrasound the kidneys are smoothly enlarged and diffusely echogenic, with a loss of corticomedullary differentiation. In utero you sometimes will not see urine in the bladder.

Neonatal Renal Vein Thrombosis - This is an associated condition of maternal diabetes. It is typically unilateral (usually left). The theory is that it starts peripherally and progresses toward the hilum. When acute, will cause renal enlargement and chronically, will result in renal atrophy.

Neonatal Renal Artery Thrombosis - This occurs secondary to umbilical artery catheters. Unlike renal vein thrombosis it does NOT present with renal enlargement but instead severe hypertension.

Congenital Ureter and Urethra

Congenital (primary') MEGAureter - This is a “wastebasket” term, for an enlarged ureter which is intrinsic to the ureter (as opposed to the result of a distal obstruction). Causes include (1) Distal adynamic segment (analogous to achalasia, or colonic Hirschsprungs), (2) reflux at the UVJ, (3) its just wants to be big (totally idiopathic). The distal adynamic type “obstructing primary megaureter” can have some hydro, but generally speaking an *absence of dilation of the collecting system* helps distinguish this from an actual obstruction.

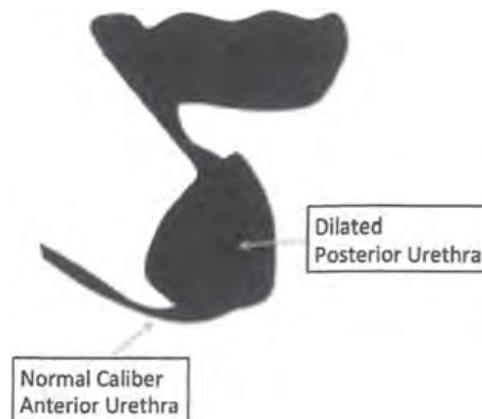
Retrocaval Ureter (circumcaval)- Although the name implies that this is the result of a maldeveloped ureter, it's actually a developmental anomaly of the I VC. Most of the time it's asymptomatic, but can cause partial obstruction and recurrent UTI. IVP will show a “reverse J” or “fishhook” appearance of the ureter.

Duplicated System - The main thing to know about duplicated systems is the so called “**Weigert-Meyer Rule**” where the upper pole inserts inferior and medially. The upper pole is prone to ureterocele formation and obstruction. The lower pole is prone to reflux. Kidneys with duplicated systems tend to be larger than normal kidneys. In girls a duplicated system can lead to incontinence (ureter may insert below the sphincter - sometimes into the vagina).

Ureterocele - A cystic dilation of the intravesicular ureter, secondary to obstruction at the ureteral orifice. IVP will show the “cobra head” sign, with contrast surrounded by a lucent rim, protruding from the contrast filled bladder. This is associated with a duplicated system (specifically the upper pole).

Ectopic Ureter - The ureter inserts distal to the external spincter in the vestibule. More common in females and associated with incontinence (not associated with incontinence in men). Ureteroceles are best demonstrated during the early filling phase of the VCUG

Posterior Urethral Valves: Congenital folds located near the posterior urethra. This is the most common cause of urethral obstruction in male infants. The valve causes back pressure and reflux that can eventually result in renal failure. It's treated with electrode fulguration. Now, this can be shown a variety of ways; it could be shown in the classic VCUG. The key finding on VCUG is an *abrupt caliber change between the dilated posterior urethra and normal caliber anterior urethra*. Another, much sneakier way to show this is with a fetal MRI. The MRI would have to show hydro in the kidney and a “key hole” bladder appearance. “Peri-renal fluid collection” is a buzzword, and it's the result of fomicel rupture. Obviously that is non-specific and can be seen with any obstructive pathology.



Vesicoureteral Reflux (VUR) - Normally the ureter enters the UVJ at an oblique angle so that a “valve” is developed. If the angle of insertion is abnormal reflux can develop. This can occur in the asymptomatic child, but is seen in 50% of children with UTIs. The recommendations for when the boy/girl with a UTI should get a VCUG to evaluate for VUR is in flux (not likely to be tested). Most of the time VUR resolves by age 5-6.

There is a grading system for VUR which goes 1-5.

- *One is reflux halfway up the ureter,*
- *Two is reflux into a non-dilated collecting system,*
- *Three you have dilation of the collecting system,*
- *Four the system gets mildly tortuous, and*
- *Five the system is very tortuous.*

A sneaky trick would be to show the echogenic mound near the UVJ, that results from injection of “deflux”, which is a treatment urologist try. Essentially, they make a bubble with this proprietary compound in the soft tissues near the UVJ and it creates a valve (sorta). Anyway, they show it in a lot of case books and text books so just keep your eyes peeled.

Additional Pearl is that chronic reflux can lead to scarring. This **scarring can result in hypertension** and/or chronic renal failure.

Congenital Bladder

The Urachus: The umbilical attachment to the bladder (started out being called the allantois, then called the Urachus). This usually atrophies into the umbilical ligament. Persistent canalization can occur along a spectrum (patent, sinus, diverticulum, cyst).

Urachal tracts are often subject to infection.



The most important piece of trivia is that when these guys get cancer, it's adenocarcinoma (90% of cases). To hint at this multiple choice test writers will often use the phrase “midline bladder structure”

Renal Masses

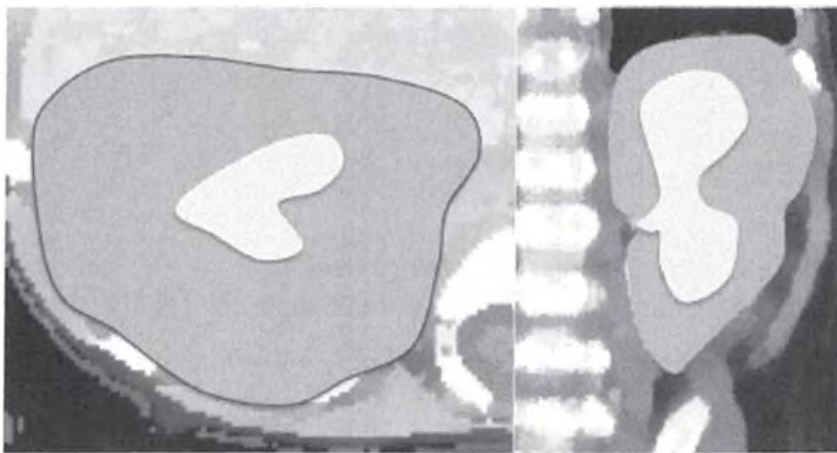
When it comes to solid renal masses - an age based strategy is the ticket.

Neonate	Around Age 4	Teenager
Nephroblastomatosis	Wilms	RCC
Mesoblastic Nephroma	Wilms Variants	Lymphoma
	Lymphoma	
	Multilocular Cystic Nephroma	

Solids Age 0-3

Nephroblastomatosis - These are persistent nephrogenic rests beyond 36 weeks. It's sorta normal (found in 1% of infants). But, can be a precursor to Wilms so you follow it. When Wilms is bilateral, 99% of the time it had nephroblastomatosis first. It goes away on its own (normally). *It should NOT have necrosis - this makes you think Wilms.* It has a variable appearance, and is often described as "homogenous." Also more commonly a focal homogenous ball, the way it's always shown in case conferences and case books is the hypodense rind.

Ultrasound screening q 3 months till age 7 is the usual routine - to make sure it doesn't go Wilms on you.



Nephroblastomatosis "Classic Look"

Mesoblastic Nephroma - "*Solid renal tumor of infancy.*" This is a fetal hamartoma, and generally benign. It is the most common neonatal renal tumor (80% diagnosed in the first month on life). Often involves the renal sinus. Antenatal ultrasound may have shown polyhydramnios.

Cystics Age 0-3

Multicystic Dysplastic Kidney You have multiple tiny cysts forming in utero. What you need to know is (1) that there is “no functioning renal tissue,” (2) contralateral renal tract abnormalities occur like 50% of the time (most commonly UPJ obstruction).

MCDK vs Bad Hydro?

- *In hydronephrosis the cystic spaces are seen to communicate.*
- *In difficult cases renal scintigraphy can be useful. MCDK will show no excretory function.*

Wilms in a 1 year old ?

*Think about associated syndromes.
Wilms loves to pal around with
Hemihypertrophy, Hyposadias,
Cryptorchidism*

I Say Beckwith-Weidemann

You Say,

- *Wilms,*
- *Omphalocele,*
- *Hepatoblastoma*

Solids Around Age 4

Wilms - “Solid renal tumor of childhood.” This is the most common solid renal tumor of childhood. This is NOT seen in a newborn. Repeat, you can NOT be born with this tumor. The average age is around 3. It typically spreads via direct invasion.

Associated Syndromes:

- *Overgrowth*
 - *Beckwith-Weidemann - Macroglossia (most common finding), Omphalocele, Hemihypertrophy, Cardiac, Big Organs.*
 - *Sotos - Macrocephaly, Retarded (CNS stuff), Ugly Face*
- *Non-Overgrowth*
 - *WAGR - Wilms, Aniridia, Genital, Growth Retardation*
 - *Drash - Wilms, Pseudohemaphroditism, Progressive Glomerulonephritis*

Wilms Variants (look just like Wilms)

- **Clear Cell** - likes to go to bones (lytic)
- **Rhabdoid** - “Terrible Prognosis” - Associated with aggressive rhabdoid brain tumors

Wilms Nevers

- **NEVER Biopsy** suspected Wilms (you can seed the tract and up the stage)
- Wilms **NEVER occurs before 2 months of age** (Neuroblastoma can)

Cystics Around Age 4

Multilocular Cystic Nephroma - “Non-communicating, fluid-filled locules, surrounded by thick fibrous capsule.” By definition these things are characterized by the absence of a solid component or necrosis. Buzzword is “protrude into the renal pelvis.” The question is likely the bimodal occurrence (4 year old boys, and 40 year old women). *I like to think of this as the Michael Jackson lesion - it loves young boys and middle aged women*

Solids in Teenagers

Renal Lymphoma and RCC can occur in teenagers. Renal lymphoma can occur in 5 year old as well. Both of these cancers are discussed in detail in the adult GU chapter.

Other GU Masses / Cancers

Rhabdomyosarcoma - This is the most common bladder cancer in humans less than 10 years of age. They are often infiltrative, and it’s hard to tell where they originate from. **“Paratesticular Mass” is often a buzzword.** They can met to the lungs, bones, and nodes. The Botryoid variant produces a polypoid mass, which looks like a bunch of grapes.

Neuroblastoma - Isn’t a Renal Mass, but is frequently contrasted with Wilms so I want to discuss it in the renal section. It is the most common extracranial solid childhood malignancy. They typically occur in very young kids (you can be born with this). 95% of cases occur before age 10. They occur in the abdomen more than the thoracic (adrenal 35%, retroperitoneum 30%, posterior mediastinum 20%, neck 5%).

Staging: Things that up the stage include crossing the midline, and contralateral positive nodes. These things make it Stage 3

Better Prognosis Seen with - Diagnosis in Age < 1, Thoracic Primary, Stage 4S.

Stage 4S: ****HIGH YIELD**

- Seen < 1 year of Age
- Distal Mets Confined to Skin, Liver, and Bone Marrow
- Excellent Prognosis

Associations:

- NF-1, Hirschsprungs, DiGeorge, Beckwith Wiedemanns
- Most are sporadic

Random Trivia:

- *Opsomyoclonus (dancing eyes, dancing feet) - paraneoplastic syndrome associated with neuroblastoma.*
- *“Raccoon Eyes ” is a common way for orbital neuroblastoma mets to present*
- *MIBG is superior to Conventional Bone Scan for Neuroblastoma Bone Mets*
- *Neuroblastoma bone mets are on the “lucent metaphyseal band Ddx ”*
- *Sclerotic Bone mets are UNCOMMON*
- *Urine Catecholamines are always (95%) elevated*

Neuroblastoma	Wilms
Age: usually less than 2 (can occur in utero)	Age: Usually around age 4 (never before 2 months)
Calcifies 90%	Calcifies Rarely (<10%)
Encases Vessels (doesn't invade)	Invades Vessels (doesn't encase)
Poorly Marginated	Well Circumscribed
Mets to Bones	Doesn't usually met to bones (unless clear cell wilms variant).

Adrenal

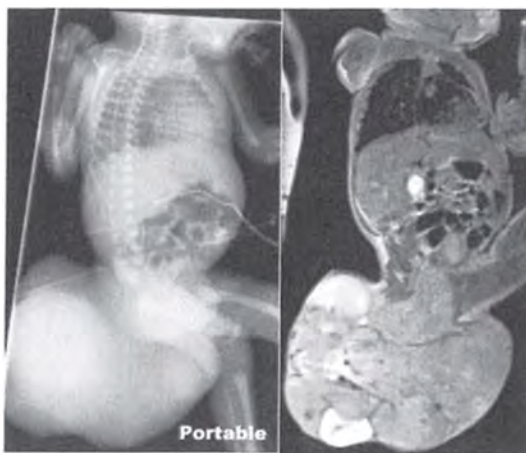
Neuroblastoma - *Discussed above in the renal section*

Neonatal Adrenal Hemorrhage - This can occur in the setting of birth trauma or stress. Not calling it a neuroblastoma is the key thing (obviously this is not surgical). Ultrasound can usually tell the difference (**adrenal hemorrhage is anechoic and avascular**, neuroblastoma is echogenic and hypervascular). MRI could also be done to problem solve if necessary (**Adrenal Hemorrhage low T2, Neuroblastoma high T2**. *The real answer is “what does it do a week later. ”* Serial ultrasound will show it decrease in size.

GYN/Pelvic

Hydrometrocolpos - Essentially the vagina won't drain the uterus. This condition is characterized on imaging by an expanded fluid filled vaginal cavity with associated distention of the uterus. You can see it presenting in infancy as a mass, or as a teenager with delayed menarche. Causes include imperforate hymen (most common), vaginal stenosis, lower vaginal atresia, and cervical stenosis. For multiple choice trivia think about this as a "midline pelvic mass", which can cause hydronephrosis (mass effect from distended uterus).

Sacroccygeal Teratoma - This is the most common tumor of the fetus or infant. These solid or cystic masses are typically large and found either on prenatal imaging or birth. They can cause mass effect on the GI system, hip dislocation, nerve compression causing incontinence, and high output cardiac failure. They are usually benign (80%). Those presenting in older infants tend to have a higher malignant potential. The location of the mass is either external to the pelvis (47%), internal to the pelvis (9%), or dumbbelled both inside and outside (34%).



Sacroccygeal Teratoma

There is another classification that discusses involvement of the abdomen. Type II does NOT extend into the abdomen, Type III does. This matters because it changes the surgical management.

Ovary - A complete discussion of ovarian masses is found in the GYN chapter. I'll briefly cover some PEDs specific ovarian issues.

Torsion: In an adult ovarian torsion is almost always due to a mass. In a child torsion can occur with a normal ovary, secondary to excessive mobility of the ovary. As described in the GYN chapter you are going to see an enlarged (swollen) ovary, with peripheral follicles, with or without arterial flow.

Masses: About two thirds of ovarian neoplasms are benign dermoids/teratomas (discussed in detail in the GYN chapter). The other one third cancer. The cancers are usually germ cells (75%). Again, mural nodules and thick septations should clue you in that these might be cancer. Peritoneal implants, ascites, lymphadenopathy, are all bad signs and would over-ride characteristics of the mass.

Scrotum

Random Scrotum Trivia

Hydrocele: Collection of serous fluid and is the most common cause of painless scrotal swelling. Congenital hydroceles result from a patent processus vaginalis that permits entry of peritoneal fluid into the scrotal sac.

Complicated Hydrocele: This is either a hematocele vs pyocele. The distinction is clinical.

Varicocele: Most of these are idiopathic and found in adolescents and young adults. They are more frequent on the left. They are uncommon on the right, and if isolated (not bilateral) should stir suspicion for abdominal pathology (nutcracker syndrome, RCC, retroperitoneal fibrosis).

HSP: this vasculitis is the most common cause of idiopathic scrotal edema (more on this in the vascular chapter).

Acute Pain in or around the Scrotum

The top three considerations in a child with acute scrotal pain are torsion of the testicular appendage, testicular torsion, and epididymo orchitis.

Epididymitis - The epididymal head is the most affected. Increased size and hyperemia are your ultrasound findings. This occurs in two peaks: under 2 and over 6. You can have infection of the epididymis alone or infection of the epididymis and testicle (isolated orchitis is rare).

Testicular Calcifications	
Tiny (micro)	Seminoma
Big	Germ Cell Tumor

Orchitis - when isolated basically only occurs from mumps.

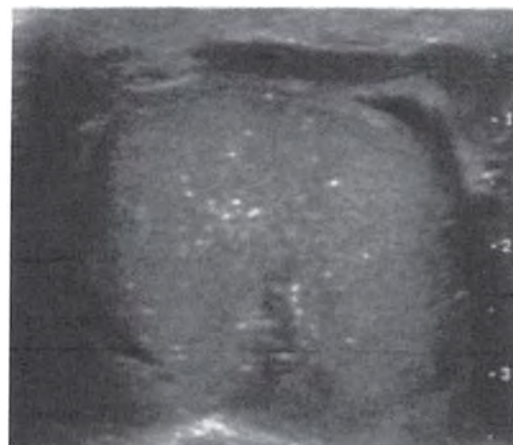
Torsion of the Testicular Appendages - This is the most common cause of acute scrotal pain in age 7-14. The testicular appendage is some vestigial remnant of a mesonephric duct. Typical history is a sudden onset of pain. **Blue Dot Sign** on physical exam (looks like a blue dot), is a classic question. Enlargement of the testicular appendage to greater than 5 mm is considered by some as the best indicator of torsion

Torsion of the Testicle - Results from the testis and spermatic cord twisting within the serosal space leading to ischemia. The testable trivia is that it is caused by a *failure of the tunica vaginalis and testis to connect* “**Bell Clapper Deformity**”. This deformity is usually bilateral, so if you twist one they will often orchiopexy the other one. If it was 1950 you’d call in your nuclear medicine tech for scintigraphy. Now you just get a Doppler ultrasound. Findings will be absent or asymmetrically decreased flow, asymmetric enlargement and slightly decreased echogenicity of the affected testis.

Testicular Masses

Testicular Masses can be thought of as intratesticular or extratesticular. With regard to intratesticular masses, ultrasound can show you that there is indeed a mass but there are no imaging features that really help you tell which one is which. **If the mass is extratesticular , the most likely diagnosis is an embryonal rhabdomyosarcoma** from the spermatic cord or epididymis

Testicular Microlithiasis - This appears as multiple small echogenic foci within the testes. Testicular microlithiasis is usually an incidental finding in scrotal US examinations performed for unrelated reasons. It might have a relationship with Germ Cell Tumors (controversial). Follow-up in 6 months, then yearly is probably the recommendation.



Microlithiasis

Testicular Cancer: The histologic breakdown is as follows:

- Germ Cell (90%)
 - Seminoma (40%) - seen more in the 4th decade
 - Non Seminoma (60%)
 - Teratoma, Yolk Sacs, Mixed Germ Cells, Etc...
- Non Germ Cell (10%)
 - Sertoli
 - Leydig

The two Germ Cell Tumors seen in the first decade of life are the yolk sac tumor, and the teratoma.

Yolk Sac Tumor: Heterogeneous Testicular Mass in < 2 year old = Yolk Sac Tumor. AFP is usually super elevated.

Teratoma - Pure testicular teratomas are only seen in young kids < 2. Mixed teratomas are seen in 25 year olds. Unlike ovarian teratoma, these guys often have aggressive biological behavior.

Choriocarcinoma: This is a aggressive highly vascular tumor, seen more in the 2nd decade.



Teratoma

Sertoli Cell Tumors - These testicular tumors are usually bilateral and are visualized on US as “bumed-out” tumors (dense echogenic foci that represent calcified scars). A subtype of Sertoli cell tumor associated with Peutz-Jeghers syndrome typically occurs in children. If they show you the Peutz-Jegher lips and bilateral scrotal masses, this is the answer.

Testicular Lymphoma - Just be aware that lymphoma can “hide” in the testes because of the blood testes barrier. Immunosuppressed patients are at increased risk for developing extranodal/ testicular lymphoma. On US, the normal homogeneous echogenic testicular tissue is replaced focally or diffusely with hypoechoic vascular lymphomatous tissue

Buzzword = multiple hypoechoic masses of the testicle.

Peds MSK

Fracture: In general little kids bend they don't break. You end up with lots of buckles and greensticks. For problem solving you can get a repeat in 7-10 days as periosteal reaction is expected in 7-10 days. Kids tend to heal completely, often with no sign of prior fracture.

Involvement of the Physis: When the physis is involved you might have some problems, including growth arrest.

Salter-Harris Classification lends itself well to multiple choice:

Type 1: S - Slipped

Complete physal fracture, with or without displacement. These are common in kids less than 5. Usually they do well.

Type 2: A - Above (or "Away from the Joint")

Fracture involves the metaphysis. This is the most common type (75%). Usually they do well

Type 3: L - Lower (3 is the backwards "E" for Epiphysis)

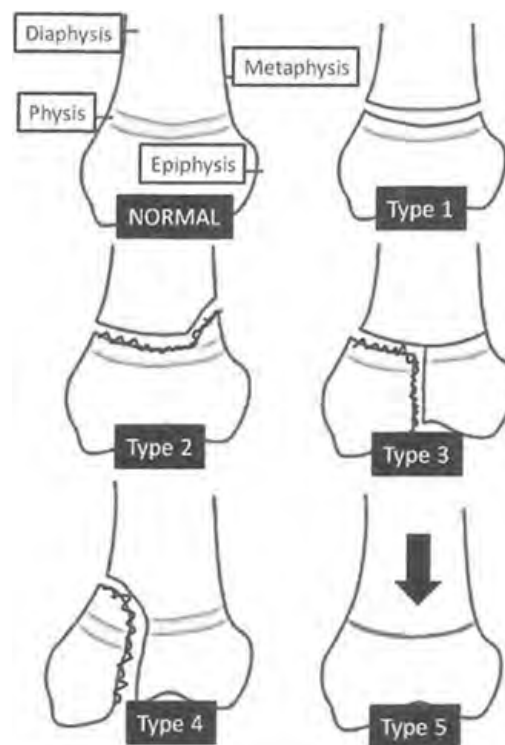
Fracture involves the epiphysis. These guys have a chance of growth arrest, and will often require surgery to maintain alignment

Type 4: T Through

Fracture involves the metaphysis and epiphysis. These guys don't do as well, often end up with growth arrest, or focal fusion. They require anatomic reduction and often surgery.

Type 5: R Ruined

Compression of the growth plate. It occurs from axial loading injuries, and has a very poor prognosis. These are easy to miss, and often found when looking back at comparisons (hopefully ones your partner read). The buzzword is **"bony bridge across physis"**.



Toddler's Fracture: Oblique fracture of the midshaft of the tibia seen in a child just starting to walk (new stress on bone). If it's a spiral type you probably should query non-accidental trauma. The typical age is 9 months - 3 years.

Stress Fracture in Children: This is an injury which occurs after repetitive trauma, usually after new activity (walking). The most common site of fracture is the tibia - proximal posterior cortex. The tibial fracture is the so called "toddler fracture" described above. Other classic stress fractures include the **calcaneal fracture - seen after the child has had a cast removed and returns to normal activity.**

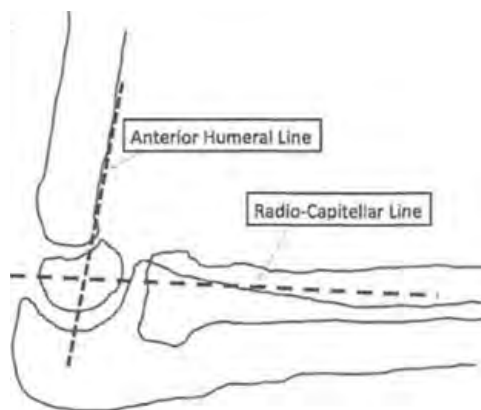
The Elbow:

My God... theseped elbows.

Every first year resident knows that elevation of the fat pad (sail sign) should make you think joint effusion and possible occult fracture. Don't forget that sometimes you can see a thin anterior pad, but you should never see the posterior pad (posterior is positive). I like to bias myself with statistics, when I'm hunting for the peds elbow fracture. The most common fracture is going to be a supracondylar fracture (>60%), followed by lateral condyle (20%), and medial epicondyle (10%).

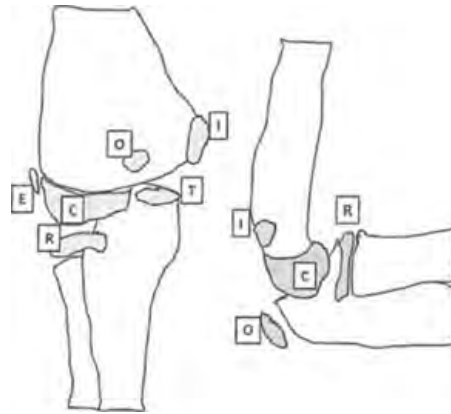
Radiocapitellar Line: This is a line through the center of the radius, which should intersect the middle of the capitellum on every view (regardless of position). If the radius is dislocated it will NOT pass through the center of the capitellum

Anterior Humeral Line: This time you need a true lateral. A line along the anterior surface of humerus, which should pass through the middle third of the capitellum. With a supracondylar fracture (the most common peds elbow fracture) you'll see this line pass through the anterior third.



Ossification Centers can be a source of trickery.

Remember they occur in a set order (CRITOE), Capitellum (Age 1), Radius (Age 3), Internal (medial epicondyle - Age 5), Trochlea (Age 7), Olecranon (Age 9), and External (lateral epicondyle - Age 11).



Peds Elbow Tricks:

Lateral Condyle Fx: This is the **second most common distal humerus fracture** in kids. Some dude named Milch classified them. The thing to know is a fracture that passes through the capitello-trochlear groove is unstable (Milch II). Since it's really hard to tell this, **treatment is based on the displacement of the fracture fragment (> or < 2mm).**

Trochlea - it can have multiple ossification centers, so it **can have a fragmented appearance.**

Medial Epicondyle Avulsion (Little League Elbow)- There are two major tricks with this one. (1) Because it's an extra articular structure, its avulsions **will not necessarily result in a joint effusion.** (2) It **can get interposed** between the articular surface of the humerus and olecranon. Avulsed fragments can get stuck in the joint, even when there is no dislocation.

Anytime you see a dislocation - ask yourself

- * *Is the patient 5 years old? And if so*
- * *Where is the medial epicondyle.*

The importance of IT (crIToe) -

- * *You should never see the trochlea, and not see the internal (medial epicondyle), if you do it's probably a displaced fragment*

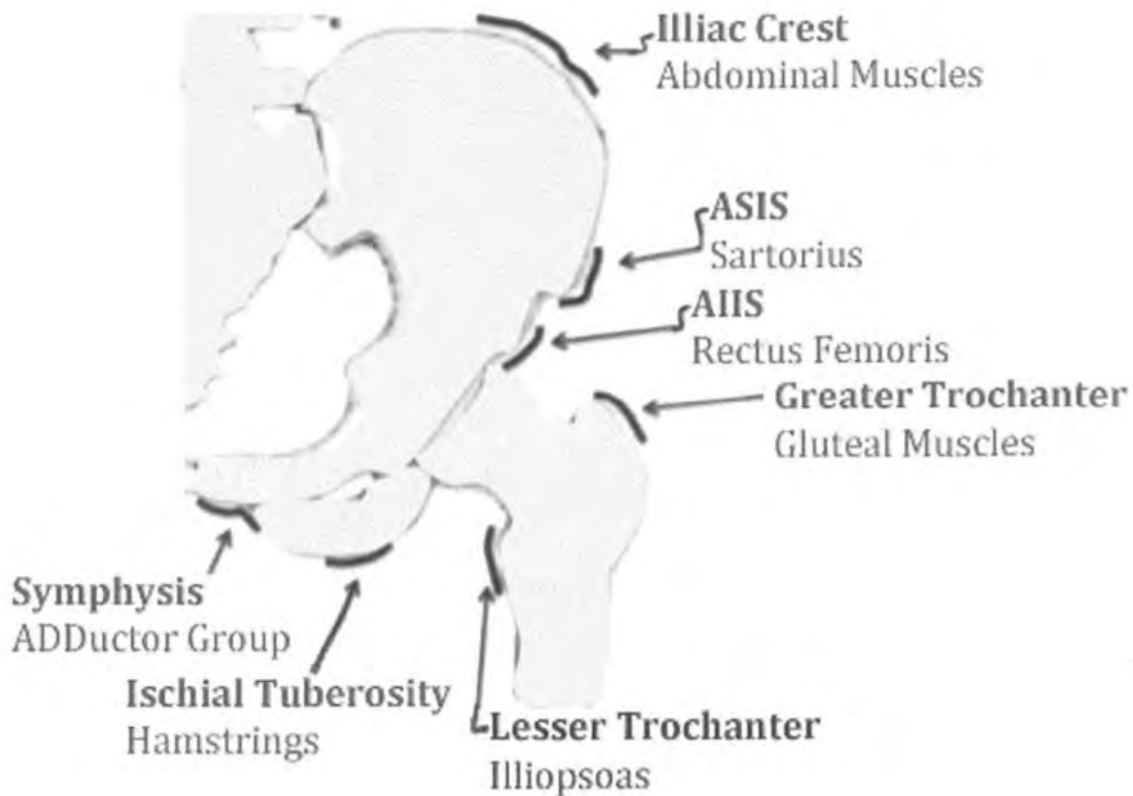
Common vs Uncommon: Don't get it twisted.

<i>Common</i>	<i>Uncommon</i>
<i>Lateral Condylar Fx</i>	<i>Lateral Epicondyle Fx</i>
<i>Medial Epicondyle Fx</i>	<i>Medial Condyle</i>

Nursemaids Elbow: When a child's arm is pulled on, the radial head may sublux into the annular ligament. X-rays typically don't help, unless you supinate the arm during lateral position (which often relocates the arm).

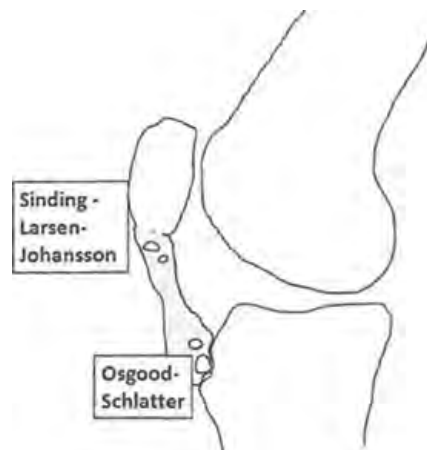
Avulsion Injuries:

Kids tendons tend to be stronger than their bones, so avulsion injuries are more common (when compared to adults). The pelvis is the classic location to test this.



Sinding-Larsen-Johansson - This is a chronic traction injury at the **insertion of the patellar tendon on the patella**. It's seen in active adolescents between age 10-14. **Kids with cerebral palsy** are prone to it.

Osgood-Schlatter - This is due to repeated micro trauma to the patellar tendon on its insertion at the tibial tuberosity. It's bilateral 25% of the time, and more common in boys.



Distal Femoral Metaphyseal Irregularity (Cortical Desmoid): This is a lucency seen along the back of the posteromedial aspect of the distal femoral metaphysis. If they show you a lateral knee x-ray, and there is an irregularity or lucency on the back of the femur this is it. It's often bilateral. Buzzwords include "Scoop like defect" with an "irregular but intact cortex."

This is a total incidental finding and is a don't touch lesion. **Don't biopsy it, Don't MRI it.** Just leave it alone. If you really want to know, it's probably a chronic tug lesion from the adductor magnus.

Blounts (tibia vara). Varus angulation occurring at the medial aspect of the proximal tibia (*varus bowing occurs at the metaphysis not the knee*). This is often bilateral, and NOT often seen before age 2 (*two sides, not before two*). Later in the disease progression the medial metaphysis will be depressed and an osseous outgrowth classically develops. You can see it in two different age groups; (a) early - which is around age 3, and (b) late - which is around age 12.



Blounts – Tibia Vara

Periosteal Reaction in the Newborn:

Congenital Rubella: Bony changes are seen in 50% of cases, with the classic buzzword being "**celery stalk**" appearance, from generalized lucency of the metaphysis. This is usually seen in the first few weeks of life.

Syphilis: Bony changes are seen in 95% of cases. Bony changes do NOT occur until 6-8 weeks of life (Rubella changes are earlier). Metaphyseal lucent bands and periosteal reaction along long bones can be seen. The classic buzzword is "**Wimberger Sign**" or **destruction of the medial portion of the proximal metaphysis of the tibia.**



Syphilis

Wimberger Sign + Periosteal Rx

Caffey Disease - Have you ever seen that giant multiple volume set of peds radiology books? Yeah, same guy. This thing is a self limiting disorder of soft tissue swelling, periosteal reaction, and irritability **seen within the first 6 months of life**. The **classic picture is the really hot mandible on bone scan**. The mandible is the most common location (clavicle, and ulna are the other classic sites). It's rare as hell, and probably not even real. There have been more sightings of Chupacabra in the last 50 years.

Prostaglandin Therapy - Prostaglandin E1 and E2 (often used to keep a PDA open) can cause a periosteal reaction. The classic trick is to show a chest x-ray with sternotomy wires (or other hints of congenital heart), and then periosteal reaction in the arm bones.

Neuroblastoma Mets - This is really the only childhood malignancy that occurs in newborns and mets to bones.

Physiologic Growth: So this is often called "Physiologic Periostitis of the Newborn", which is totally false and wrong. It **does NOT happen in newborns**. *You see this around 3 months of age*, and it should resolve by six months. **Proximal involvement (femur) comes before distal involvement (tibia)**. It **always involves the diaphysis**.

*It is **NOT** physiologic periostitis if:*

- * You see it before 1 month*
- * You see it in the femur before the tibia*
- * It does not involve the diaphysis.*

Abuse - Some people abuse drugs, some just can't stand screaming kids, some suffer both shortcomings. More on this later.

Other "Aggressive Processes" in Kids

Langerhans Cell Histiocytosis (LCH) - Also known as EG (eosinophilic granuloma). It's twice as common in boys. Skeletal manifestations are highly variable, but let's just talk about the classic ones: *

- * **Skull - Most common site**. Has **"beveled edge"** from uneven destruction of the inner and outer tables. If you see a round lucent lesion in the skull of a child think this (and neuroblastoma mets).
- * **Ribs** - Multiple lucent lesions, with an expanded appearance
- * **Spine - Vertebral plana**

Ewing Sarcoma, and **Osteosarcoma** are covered in depth in the MSK chapter

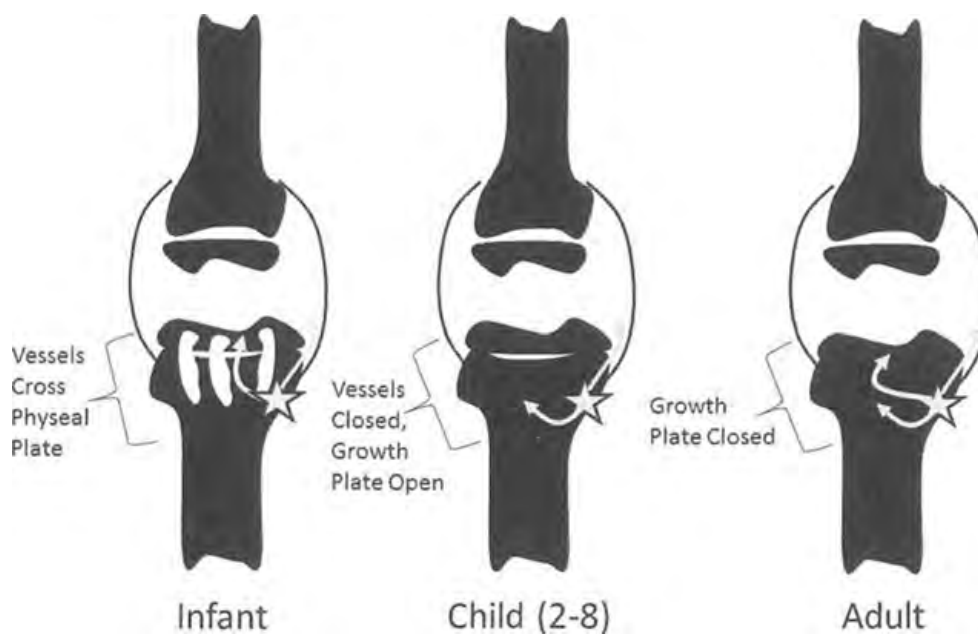
Osteomyelitis - It usually occurs in babies (30% of cases less than 2 years old). It's usually hematogenous (adults it directly spreads).

There are some changes that occur over time, which are potentially testable.

Newborns - They have open growth plates and perforating vessels which travel from the metaphysis to the epiphysis. Infection typically starts in the metaphysis (it has the most blood supply because it is growing the fastest), and then can spread via these perforators to the epiphysis.

Kids - Later in childhood, the perforators regress and the avascular epiphyseal plate stops infection from crossing over. This creates a "septic tank" scenario, where infection tends to smolder. In fact, 75% of cases involve the metaphyses of long bones (femur most common).

Adults - When the growth plates fuse, the bander of an avascular plate is no longer present, and infection can again cross over to the epiphysis to cause mayhem.



Trivia to know:

- Hematogenous spread more common in kids (direct spread in adult)
- Metaphysis most common location, with target changes as explained above
- Bony changes don't occur on x-ray for around 10 days.
- It's serious business and can cause serious deformity if it spreads to involve the joint.

Skeletal Dysplasia

There is a bunch of vocabulary to learn regarding dysplasias:

English	Fancy Doctor Speak
Short Fingers	Brachydactyly
Too Many Fingers	Polydactyly
Two or More Fused Fingers (<i>“Sock Hand” -I call it</i>)	Syndactyly
Contractures of Fingers	Camptodactyly
Inclinded Fingers (Usually 5 th)	Clinodactyly
Long, Spider-Like Fingers	Arachnodactyly
Limb is Absent	Amelia
Limb is mostly Absent	Meromelia
Hands / Feet (distal limbs) are Short	Acromelic
Forearm or Lower Leg are short (middle limbs)	Mesomelic
Femur or Humerus (proximal limbs) are short	Rhizomelic
Short All Over	Micromelic

How I remember the lengths: Meso is in the middle, Aero sounds like acromegaly (they get big hands), and the other one is the other one (Rhizo).

There are tons of skeletal dysplasias and extensive knowledge of them is way way way way beyond the scope of the CORE. Instead I'm going to mention 3 dwarfs, and a few other miscellaneous conditions.

Dwarfs:

Achondroplasia - This is the most common skeletal dysplasia. It results from a fibroblast growth factor receptor problem (most dwarfisms do). It is a rhizomelic (short femur, short humerus) dwarf. They often have big heads, trident hands (3rd and 4th fingers are long), narrowing of the interpedicular distance, and the tombstone pelvis. Advanced paternal age is a risk factor.

Thanatophoric - This is the **most common lethal dwarfism**. They have **rhizomelic shortening** (humerus, femur). The femurs are sometimes called **telephone receivers**. They have short ribs and a long thorax, and small iliac bones. The **vertebral bones are flat** (platyspondyly), and the **skull can be cloverleaf shaped**.



Asphyxiating Thoracic Dystrophy (Jeune) - This is usually fatal as well. The big finding is the “Bell shaped thorax” with short ribs. 15% will have too many fingers (polydactyly). If they live, they have kidney problems (chronic nephritis). You can differentiate a dead Thanatophoric dwarf, from a dead Jeune dwarf by looking at their vertebral bodies. The Jeune bodies are normal (the thanatophorics are flat).

Additional Dwarf pearls;

- Ellis-Van Crevald** is the dwarf with multiple fingers.
- Pseudoachondroplasia** is this weird thing not present at birth, and spares the skull.
- Pyknodysostosis** is osteopetrosis, in a dwarf with a wide angled jaw, and osteoarcolysis.

Misc Conditions

Osteogenesis Imperfecta - They have a collagen defect and make brittle bones. Depending on the severity it can be totally lethal or more mild. It's classically shown with a **totally lucent skull**, or **multiple fractures with hyperplastic callus**. Another classic trick is to show the legs with the **fibula longer than the tibia**. They have **wormian bones**, and often flat or beaked vertebral bodies. Other trivia is the blue sclera, hearing impairment (**otosclerosis**), and that they tend to suck at football.

Osteopetrosis - They have a defect in the way osteoclasts work, so you end up with disorganized bone that is sclerotic and weak (prone to fracture). There are a bunch of different types, with variable severity. The infantile type is lethal because it takes out your bone marrow. With less severe forms, you can have abnormal diminished osteoclastic activity that varies during skeletal growth, and results in alternating bands of sclerosis parallel to the growth plate. Most likely the way this will be shown is the "bone-in-bone" appearance in the vertebral body or carpals. Picture frame vertebrae is another buzzword. Alternatively, they can show you a diffusely sclerotic skeleton, with diffuse loss of the corticomedullary junction in the long tubular bones.



Pycnodysostosis - Osteopetrosis + Wormian Bones + Acro-Osteolysis. They also have "wide (or obtuse) angled mandible", which apparently is a buzzword.



Klippel Feil - You get **congenital fusion of the cervical spine** (sorta like JRA). The cervical vertebral bodies will be tall and skinny. There is often a Sprengel deformity (high riding scapula). Another common piece of trivia is to show the omovertebral bone - which is just some big stupid looking vertebral body.

Hunters /Hurlers / Morquio: All three of these are mucopolysaccharidoses. Findings include **oval shaped vertebral bodies with anterior beak**. The beak is actually mid in morquio, and inferior in hurlers. **Clavicles and ribs are often thick (narrow more medially) - like a canoe-paddle**. The pelvis shape is described as the opposite of achondroplasia - the iliac wings are tall and flaired. The hand x-ray is the most commonly shown in case books and gives you **wide metacarpal bones with proximal tapering**.

Few More Trivia points on Morquio:

- They are dwarfs
- The most common cause of death is cervical myelopathy at C2
- The bony changes actually progress during the first few years of life

Gauchers - This is the most common lysosomal storage disease. It gives you a big spleen, and big liver among a few bone signs.

*A VN of the Femoral Hips

*H Shaped Vertebra

* Bone Infarcts (lots of them)

* Erlenmeyer Flask Shaped Femurs

Caudal Regression Syndrome - This is a spectrum that includes sacral and or coccyx agenesis. You see it with VACTERL and Currarino Triads Syndromes.

Scoliosis - Lateral curvature of the spine, which is usually idiopathic in girls. It can also be from vertebral segmentation problems. NF can cause it as well (that's a piece of trivia).

Radial Dysplasia- Absence or hypoplasia of the radius (usually with a missing thumb) is a differential case (VACTER, Holt-Oram, Fanconi Anemia, Thrombocytopenia Absent Radius). As a point of trivia TAR kids will have a thumb.

Neurofibromatosis - Just briefly remember that type 1 can cause **anterior tibial bowing**, and **pseudo-arthrosis at the distal fibula**. This is an Aunt Minnie. You can also get scoliosis.

Hand Foot Syndrome - The classic history is hand or foot pain / swelling in an infant with sickle cell. This is a dactylitis, and felt to be related to ischemia. It will resolve on its own, after a few weeks. Radiographs can show a periostitis two weeks after the pain goes away.



NF -1 Anterior Bowing

Feet

Congenital foot is a complicated and confusing topic, about which i will avoid great detail because it is well beyond the scope of the CORE. For me, vocabulary is essential for understanding what was going on.

- * *Talipes = Congenital,*
- * *Pes = Foot or Acquired*
- * *Equines = “Plantar Flexed Ankle ”, Heel Cord is often tight, and the heel won’t touch the floor*
- * *Calcaneus = Opposite of Equinus. The Calcaneus is actually angled up*
- * *Varus = Forefoot in*
- * *Valgus = Forefoot out*
- * *Cavus = High Arch*
- * *Planus = Counter part of Cavus -flat foot*
- * *Supination - Inward rotation - “Sole of foot in ”*
- * *Pro nation - Outward rotation — “Sole offoot out”*

The next concept is that the talus is fixed to the leg, and that rotation is described relative to it.

Flat Foot (Pes Planus)-This can be congenital or acquired. The peds section will cover congenital and the adult MSK section will cover acquired. The congenital types can be grouped into flexible or rigid (the flexible types are more common in kids). The distinction can be made with plantar flexion views (flexible improves with stress). The ridged subtypes can be further subdivided into tarsal coalition and vertical talus.

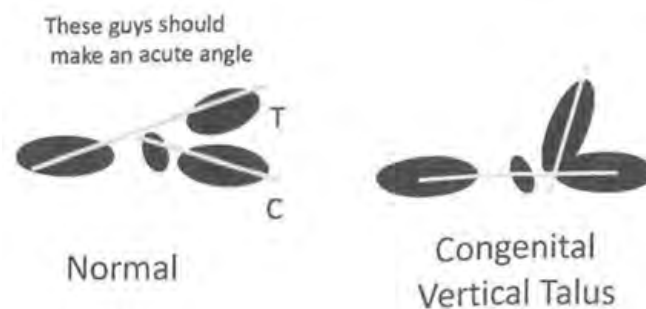
Trivia to know: Hindfoot valgus, with talocalcaneal angle less than 35 degrees.

Tarsal Coalition - There are actually several subtypes of this, with the calcaneo-navicular being the most common, and the talus to the calcaneus being the second most common. Up to 50% are bilateral. You can have bony or fibrous/cartilaginous subtypes. The fibrous/cartilaginous types are more common than the bony types.

Calcaneonavicular - Occurs at the middle facet. Has the “continuous C-sign” and “absent middle facet sign” on the lateral view. Talar beaking (spur on the anterior talus) is also seen in about 25% of cases.

Talocalcaneal - Occurs at the anterior facet. Has the “anteater sign”

Vertical Talus (equinus hindfoot valgus) - This is sometimes called the “rocker-bottom foot” because the talus is in extreme plantar flexion with dorsal dislocation of the Navicular - resulting in a locked talus in plantar flexion. As a point of trivia this is often associated with myelomeningocele.

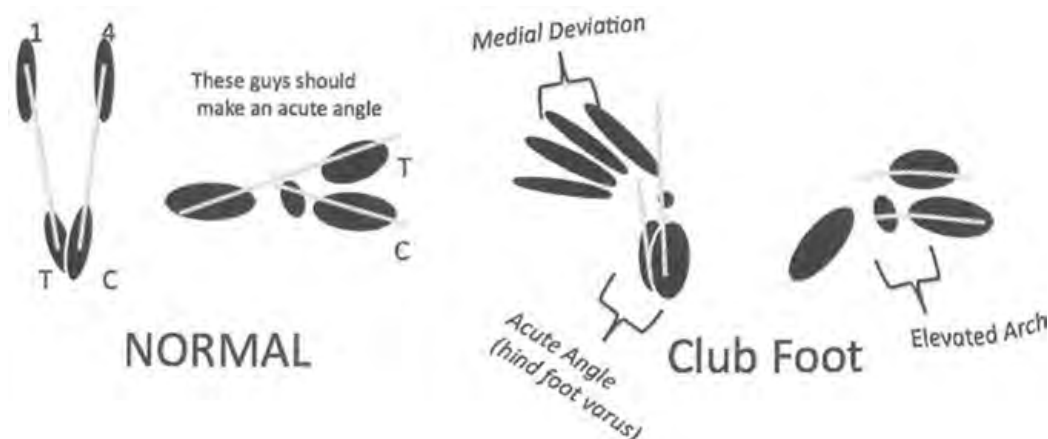


Club Foot (Talipes Equino Varus)- Translation - *Congenital Plantar Flexed Ankle Forefoot*. This is sorta why I lead with the vocab, all the congenital feet can be figured out based on the translated language. This thing is more common in boys, and bilateral about half the time. The toes are pointed down (equines), and the talocalcaneal angle is acute (varus).

Key features:

- * Hindfoot varus (decreased talocalcaneal angle)
- * Medial deviation and inversion of the forefoot
- * Elevated Plantar Arch

Trivia: The most common surgical complication is over correction resulting in a « bottom” flat foot deformity. WC & r

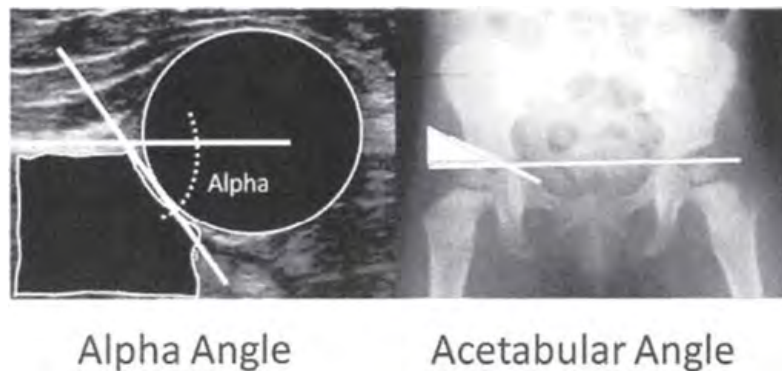


Hip Dysplasia

Developmental Dysplasia of the Hip - This is seen more commonly in females, children born breech, and oligohydramnios. The most common signs/symptoms buzzwords are asymmetric skin or gluteal folds, leg length discrepancy, palpable clunk, or delayed ambulation. It's bilateral about 1/3 of the time. Ultrasound is done to evaluate (after physical exam), and is excellent until the bones ossify (then you need x-rays). Having said that, laxity immediately after birth maybe due to maternal estrogen, and measurements made in the first week of life may not be accurate.

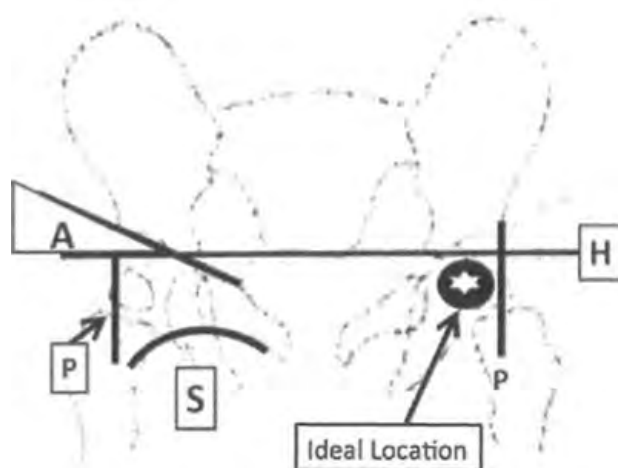
Angles:

On ultrasound the **alpha angle, should be more than 60 degrees**. Anything less than that and your cup is not deep enough to hold your ball. The plain film equivalent in the **acetabular angle, which is the complimentary angle (and therefore should be less than 30)**.



The acetabular angle should decrease from 30 degrees at birth to 22 degrees at age 1. DDH is the classic cause of an increased angle, but neuromuscular disorder can also increase it.

The position of the femoral epiphysis (or where it will be) should be below Hilgenreiner's line "H", and medial to Perkin's Line "P". Sheeton's Line "S" should be continuous.



Proximal Focal Femoral Deficiency - This is a congenital zebra, which ranges from absent proximal femur to hypoplastic proximal femur. You get a varus deformity. This is a mimic of DDH, but DDH will have normal femur leg length.

Septic Arthritis- This is serious business , and considered the most urgent cause of painful

hip in a child. Wide joint space (lateral displacement of femoral head), should prompt an ultrasound, and that should prompt a joint tap. If you have low suspicion and don't want to tap the hip, You could pull on the leg under fluoro and try and get gas in the joint. This air arthrogram sign supposedly excludes a joint effusion (and therefore a septic joint) - depending on who you ask.



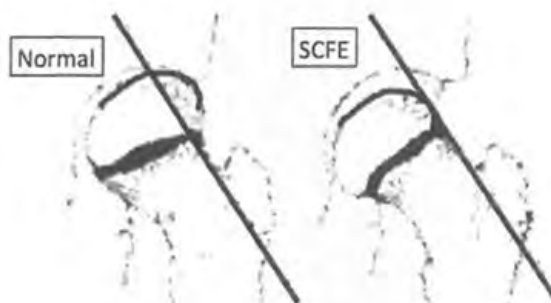
Legg-Calve-Perthes - This is AVN of the proximal femoral epiphysis. It's seen more in boys than girls (4:1), and favors **white people around age 5-8**. This is **bilateral about 10% of the time**. The subchondral lucency (crescent sign) is best seen on a frog leg. Other early signs include an asymmetric small ossified femoral epiphysis. MRI has more sensitivity. The sequella of a collapsed femoral head is easier to see.

<i>Perthes</i>	<i>SCFE</i>
<i>White Kids</i>	<i>Overweight Black Kids</i>
<i>Age 5-8</i>	<i>Age 12-15</i>
<i>Bilateral 10%</i>	<i>Bilateral 30%</i>

Slipped Capital Femoral Epiphysis

(SCFE)- This is a **type 1 salter harris**, through the proximal femoral physis. What makes this unique is that unlike most SH 1 s, this guy has a bad prognosis if not fixed. The classic history is **fat African American adolescent (age 12-15)** with hip pain. It's bilateral in 1/3 of cases (both hips don't usually present at same time). The **frog leg view is the money - this is always**

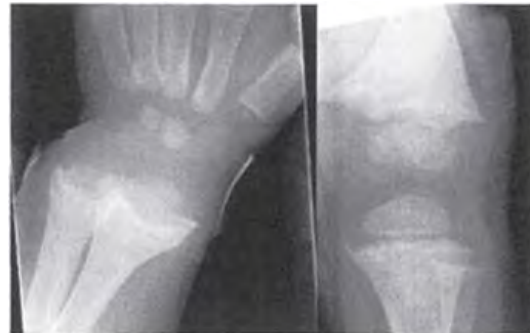
the answer on next step questions. "Klein's Line" is drawn along the edge of the femur and should normally intersect with lateral superior femoral epiphysis. This line is used to evaluate for SCFE. When the line doesn't cross the lateral epiphysis think SCFE. This is easier to do on a frog leg.



Trivia on treatment: They **pin these to stop further slippage, but they do NOT move the epiphysis back** into place (supposedly that causes AVN and chondrolysis).

Metabolic

Rickets - Not enough vitamin D. Affects the most rapidly growing bones (mostly knees and wrists). Buzzwords “**fraying, cupping, and irregularity along the physeal margin.**” They are at increased risk for SCFE. “Rachitic rosary” appearance from expansion of the anterior rib ends at the costochondral junctions. As a pearl, **rickets is never seen in a newborn** (Mom’s vitamin D is still doing its thing).



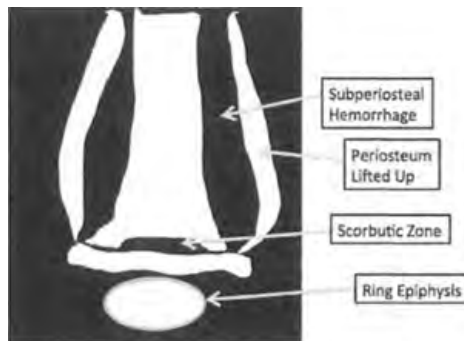
Rickets

- Fraying, cupping, and irregularity of the physeal margin

Hypophosphatasia - This looks like **Rickets in a newborn**. They will have frayed metaphyses and bowed long bones. The underlying pathology is a deficient serum alkaline phosphatase. There is variability in severity with lethal perinatal / natal forms, and more mild adult forms.

Scurvy - Not enough vitamin C. This is rare as hell outside of a pirate ship in the 1400s. For the purpose of trivia (which multiple choice tests love) the following stuff is high yield:

- * *Does NOT occur before 6 months of age (maternal stores buffer)*
 - o Bleeding Disorders Common*
 - o Subperiosteal hemorrhage (lifts up the periosteum)*
- * *Hemarthrosis*
- * *“Scorbutic rosary” appearance from expansion of the costochondral junctions (very similar to rickets).*



Scurvy

Lead Poisoning - This is most commonly seen in kids less than two who eat paint chips. The classic finding is a **wide sclerotic metaphyseal line (lead line)**, in an area of rapid growth (knee). It will not spare the fibula (as a normal variant line might).

Lucent Metaphyseal Bands - This is a classic peds DDx.

- Leukemia
- Infection (TORCH)
- Neuroblastoma Mets
- Endocrine (rickets, Scurvy)



Non-Accidental Trauma (NAT)

“Some People Just Can’t Take Screaming Kids.” Any suspicious fracture should prompt a skeletal survey (“baby gram” does NOT count). Suspicious fractures would include highly specific fractures (metaphyseal corner fracture, posterior rib fractures), or fractures that don’t make sense - toddler fracture in a non-ambulatory child.

Posterior Medial Rib Fracture: In a child under the age of 3, this is pretty reliable. Supposedly this type of fracture can only be made from squeezing a child.

Metaphyseal Corner Fractures: When this is present in a non-ambulatory patient (infant) it is HIGHLY specific. The only exception is obstetric trauma. After age 1, this becomes less specific.

Skull Fracture: The general idea is anything other than a parietal bone fracture (which is supposedly seen more with an actual accident) is concerning.

Dating the Fracture: Periosteal reaction from an injury typically means the fracture is less than a week old. Complete healing occurs in around 12 weeks. Metaphyseal, skull, and costochondral junction fractures will often heal without any periosteal reaction.

Mimics: Rickets and OI, can have multiple fractures at different sites and are the two most commonly described mimics. Wormian bones and bone mineral density issues should be identified and described for medical legal issues.

Solid Organ and Lumen Injury - Don’t forget about this as a presentation for NAT.

Duodenal hematoma and pancreatitis (from trauma) in an infant - should get you to say NAT. Just think “belly trauma in a kid that is too young to fall on the handle bars of their bike”.

Peels Neuro

Brain tumors and developmental anomalies are discussed in detail in the Neuro Chapter.

This section of the pediatric chapter will focus briefly on head ultrasound, and some random head and neck conditions.

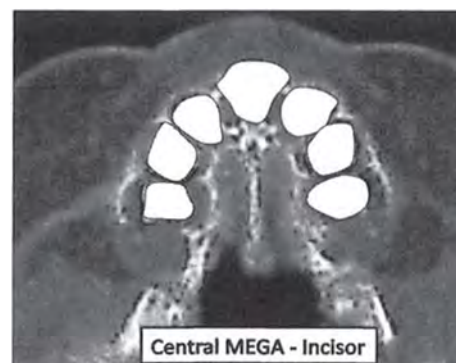
Peds Head and Neck:

Choanal Atresia - This results from the persistence of a primitive oronasal membrane that separates the nasal cavity from the oral cavity. It can be unilateral or bilateral (symptomatic immediately if bilateral). Classic story is “can’t pass NG tube.” The most important imaging finding is thickening of the vomer. It’s associated with CHARGE syndrome

CHARGE

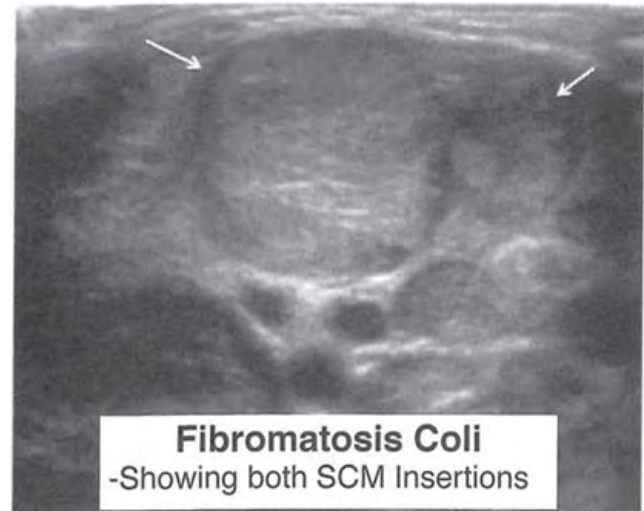
- * *Coloboma of the eye,*
- **Heart defects,*
- **Atresia of the choanae,*
- **Retardation of growth and/or development,*
- * *Genital and/or urinary abnormalities,*
- **Ear abnormalities/deafness.*

Congenital Piriform Aperture Stenosis - This results from abnormal development of the medial nasal eminences, and subsequent **failure of formation of the primary palate**. The piriform aperture of the nasal cavity is stenotic (as the name suggests), and the palate is narrow. The classic picture is the associated **central maxillary “MEGA-incisor.”** Midline defects of the brain (corpus callosal agenesis, and holoprosencephaly) are associated.



Branchial Cleft Cyst - There are several types but by far the **most common is a 2nd Branchial Cleft Cyst (95%)**. The **angle of the mandible** is a classic location. They can get infection, but are often asymptomatic. Extension of the cyst between the ICA and ECA (**notch sign**) just above the carotid bifurcation is pathognomonic.

Fibromatosis Coli - This is a **benign mass in the sternocleidomastoid** in neonates who present with torticollis (chin points towards the opposite side). Ultrasound can look scary, until you realize it's just the enlarged SCM. Sometimes it looks like there are two of them, but that's because the SCM has two heads. It goes away on its own, sometimes they do passive physical therapy.

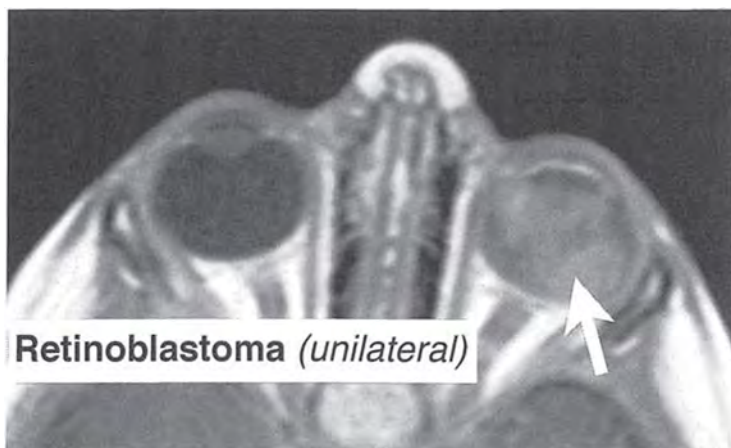


Orbital Calcifications:

Less than 3	Older than 3
Retinoblastoma	Toxo
CMV	Retinal Astrocytoma
Colobomatous	

Retinoblastoma: Discussed in detail in the neuro chapter. Calcifications are present in like 90% of cases. You can have bilateral disease, (and trilateral - involving the pineal gland with a pineoblastoma). The common thinking is the unilateral ones are sporadic, and the bilateral (and trilateral) ones are autosomal dominant related to chromosome 13. The RB suppressor on 13 links up with lots of other tumors (melanoma, fibrosarcoma, osteosarcoma, etc..).

Bad JaJu Chromosome 13



Peds Spine

Low lying cord / Tethered cord: Abnormal attachments that limit the movement of the cord within the canal. Because the canal grows faster than the cord, a fixed attachment results in cord stretching and subsequent ischemia. This can be primary (isolated), or secondary (associated with myelomeningocele, filum terminale lipoma, or trauma).

Imaging Features: Low conus (below L2), and thickened filum terminale (> 2mm).

A common piece of trivia used as a distractor is that meningocele is associated with Chiari malformations, lipomyelomeningocele is NOT.

High Yield Trivia

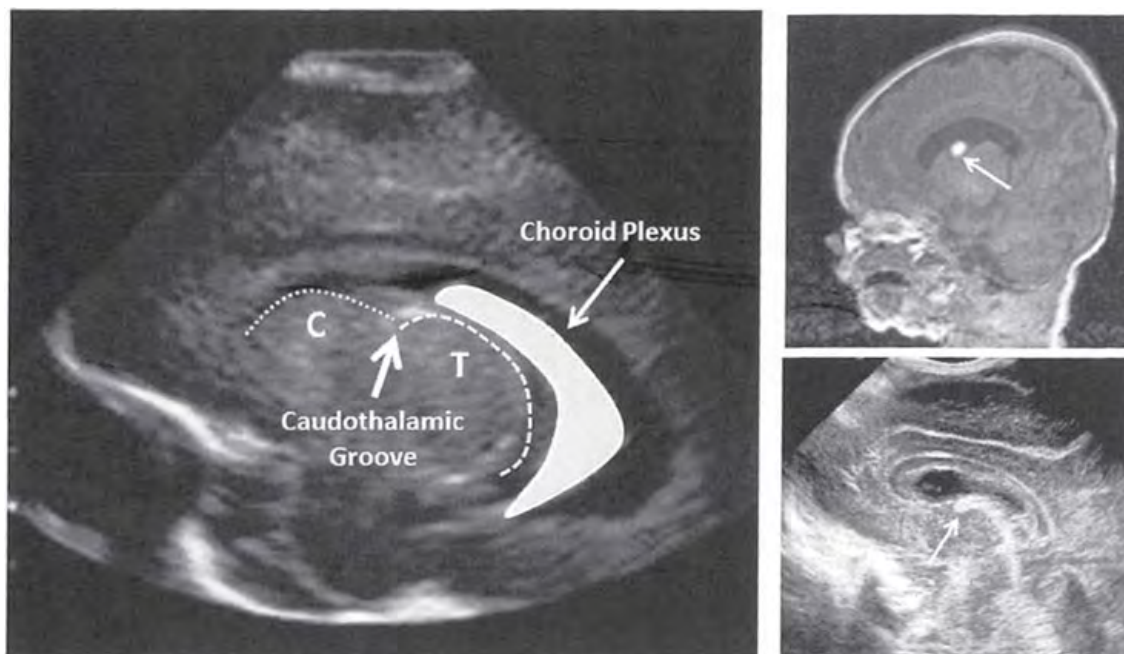
- * *Anal Atresia = High Risk For Occult Cord Problems (including tethering) - should get screened*
- * *Low lying / tethered cords are closely linked with Spina Bifida (tufts of hair)*
- * *Low Dimples (below the gluteal crease) Do NOT need screening, High Dimples Do (above the gluteal crease)*

For screening purposes just remember low dimples (below the butt crease) don't get screened, but basically everyone else does. Therefore, the question would most likely be a "which of the following does not get screened?" (answer = low dimple).

Brain

Periventricular Leukomalacia: This is the result of an ischemic / hemorrhagic injury, typically from a hypoxic insult during birthing. The kids who are at risk for this are premature, and little (less than 1500g). It favors the watershed areas (characteristically the white matter dorsal and lateral to the lateral ventricles). This will eventually cavitate into periventricular cystic changes, which occurs around 1-3 weeks post injury. Honestly, until they start to cavitate, you can't see it well, ultrasound just isn't sensitive early on. About 50% of kids with this will develop cerebral palsy.

Germinal Matrix Hemorrhage: This is seen in premature infants. By 32 weeks germinal matrix is only present at the caudothalamic groove. By 36 weeks, you basically can't have it (*if no GM then no GMhemorrhage*). The earlier they are born the more common they are. Up to 40% occur in the first 5 hours, and most have occurred by day 4 (90%). There is a grading system (1-4).



Grade 1 GM Hemorrhage
- Blood in the CT Groove

Things to know:

- Can't occur after 36 weeks
- The more premature you are, the greater the risk
- Grade 1=just blood in the caudothalamic groove
- Grade 2 = blood into NORMAL sized ventricles
- Grade 3 (hydrocephalus) and grade 4 (parenchymal bleeding) have a shitty prognosis
- Grade 1 and 2 do fine

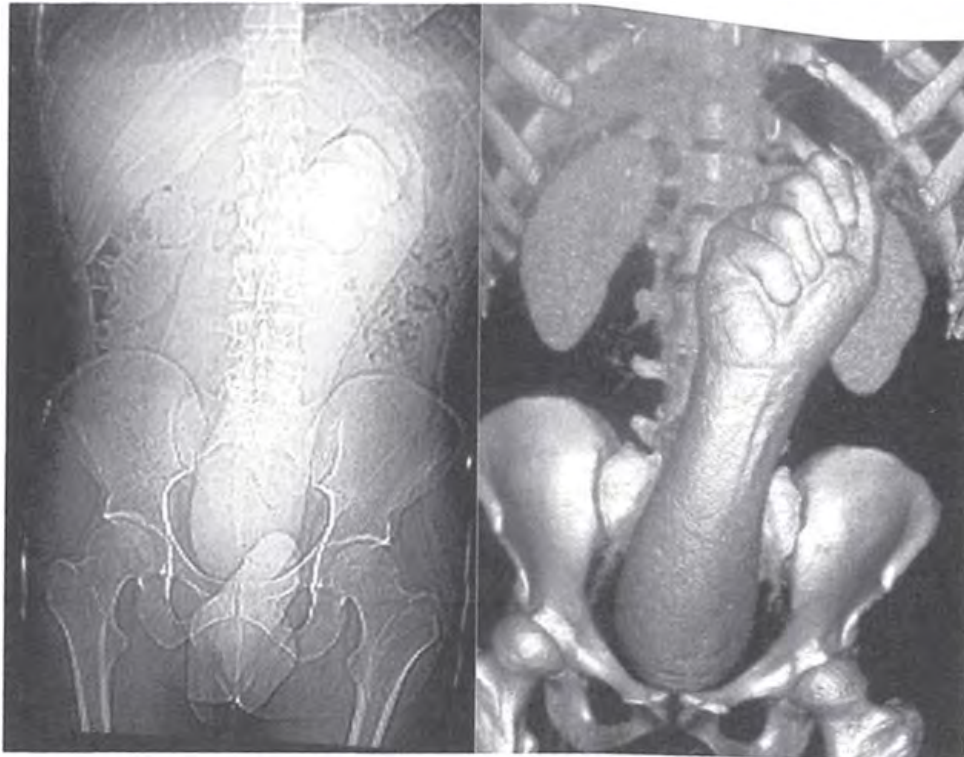
Premature Suture Closure Buzzwords:	
Sagittal	Dolichocephaly (long and skinny)
Metopic	Trigonocephaly (pointed forehead)
Coronal	Brachycephaly (also gets orbit issues “harlequin-eye)
Unilateral Lambdoid	Plagiocephaly
Bilateral Lambdoid	Turricephaly

For additional details of Congenial Brain Disorders please refer to the detailed discussion in the Neuro chapter (*found in volume 2*).

2

Gastrointestinal

Prometheus Lionhart, M.D.



GI pathology lends itself nicely to direct comparison (hepatic masses, pancreatic masses, fold pattern on barium, etc...). The differences between these entities make multiple choice test question writing fairly easy. Also, just keep in the back of your mind that more than any other section, the person writing GI questions likely trained during the Cretaceous period (*expect barium, ultrasound, and a lot of axial only CT images*).

Highest yield topics:

- Pathology shown with barium
- Vascular impression on the esophagus
- Crohns vs UC
- Hepatic Masses
- Pancreatic Masses / Cysts
- Heterotaxia Syndromes

The GI Tract

The reality is that the GI tract is best evaluated with an endoscope, with only few exceptions. The next best option is probably CT Enterography. However, for the purpose of multiple choice tests I expect the majority of GI related questions to be barium related.

Esophagus

Anatomy.

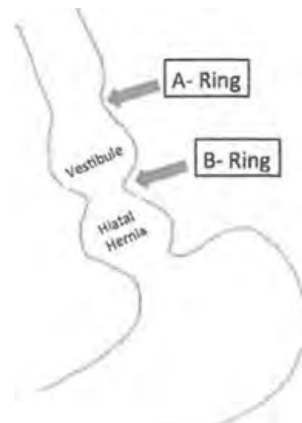
A Ring: The muscular ring above the vestibule.

B Ring: The mucosal ring Below the vestibule. This is a thin constriction at the EG junction. Dysphagia can happen if it's <13mm in diameter. **If it's narrowed you call it a Schatzki.**

Z Line: Represents the squamocolumnar junction (boundary between esophageal and gastric epithelium). This doesn't necessarily correspond with the B-ring. This is an endoscopy finding, and is only rarely seen as a thin serrated line.

Mucosa should have thin, parallel uniform folds.

Esophagus is described as a "cervical" and "thoracic" esophagus. A common piece of trivia is that the **start of the esophagus is below the cricopharyngeus muscle**, which represents the true upper esophageal sphincter and the boundary between the cervical esophagus and the pharynx.



Pathology:

Reflux Esophagitis: Causes *fold thickening*. Treated with meds (PPIs, and H2 blockers). Surgery (fundoplication) can be performed if drugs fail. If the patient has a non-reducible hernia, they may require a Collis gastroplasty (lengthening + fundoplication).

Eosinophilic Esophagitis: Classically a young man with a long history of dysphagia (and atopia, and peripheral eosinophilia). Barium shows **concentric rings** (distinct look). They fail treatment on PPIs, but get better with steroids.



Candidiasis: Discrete plaque-like lesions are the most common finding. Additional findings include: nodularity, granularity, and fold thickening may occur as a result of mucosal inflammation and edema. When it is most severe, it is manifested as a shaggy, irregular luminal surface.

Glycogen Acanthosis: This is a mimic of candidiasis, which has multiple elevated nodules in an **asymptomatic elderly patient**.

Ulcers:

- Herpes Ulcer: Small and multiple with a halo of edema (Herpes has a Halo)
- CMV and HIV: Large Flat Ulcer (they look the same)

Barretts: The way this will be shown is a **high stricture with an associated hiatal hernia**. Buzzword is **reticular mucosal pattern**.



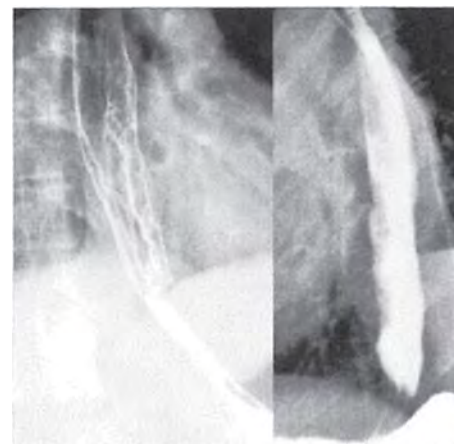
Cancer: On barium you want to see the words “*irregular contour*”, and “*abrupt (shouldered) edges*. ” The Radiologists job is to distinguish between stage 3 (Adventitia) and stage 4 (invasion into adjacent structures). I like to use stereotypes to remember the subtypes and associations:

- *Squamous*: This is a black guy who drinks and smokes, and once tried to kill himself with an alkaloid ingestion (drank lye). The stricture/ulcer/mass is in the mid esophagus.
- *Adeno*: This is a white guy, who is stressed out all the time. He has chronic reflux (history of PP1 use). He had a scope years ago that showed Barretts, and he did nothing. The stricture/ulcer/mass is in the lower esophagus.

Varices: Linear often serpentine, filling defects causing a scalloped contour. The differential diagnosis for varices includes varicoid carcinoma (this is why you need them distended on the study).

This vs That: Uphill Varices vs Downhill Varices

Uphill Varices	Downhill Varices
Caused by Portal Hypertension	Caused by SVC obstruction (catheter related, or tumor related)
Confined to Bottom Half of Esophagus	Confined to Top Half of Esophagus



Esophageal Varices
Varicoid appearance that distends / flattens out with a good barium bolus

Esophageal (enteric) duplication cysts: If they show one of these it will be on CT (what? GI path not on barium?). Seriously, they would have to show this on CT. It is gonna be in the *posterior mediastinum*, and have an ROI showing water density. This is the only way you can show this. They are benign. *Likely question is that the ileum is the most common location (esophagus is #2)*. If they are big they will present as an infant with dysphagia / breathing problems.

Lateral pharyngeal pouches: This is an Aunt Minnie situation - *protrusion of lateral pharyngeal wall*. They arise from a “weak area” of the thyrohyoid membrane which does not contain a muscular covering - the site of penetration of the laryngeal nerve. They are common, and can get big in glassblowers or wind instrument players.

Zenker Diverticulum: Diverticulum in the back (Z is in the back of the alphabet). The question they always ask is: site of weakness = **Killian Dehiscence** or triangle. Another sneaky point of trivia is that the diverticulum arise from the hypopharynx (not the cervical esophagus).

Killian-Jamieson Diverticulum: This one is **anterior and lateral**. It protrudes through an area of weakness below the attachment of the cricopharyngeus muscle on the cricoid cartilage and lateral to the suspensory ligaments of the esophagus inserting on the cricoid cartilage. This one is in the cervical esophagus.

Traction Diverticulum: Mid esophageal, and often triangular in shape.

This vs That: **Traction vs Pulsion Diverticulum**

Traction	Pulsion
Triangular	Round
Will Empty	Will NOT Empty (contain no muscle in their walls)



Epiphrenic Diverticula: Located just above the diaphragm (**usually on the right**). They are considered pulsion types (associated with motor abnormality).

Esophageal Pseudodiverticulosis: This is an Aunt Minnie. What you have are dilated submucosal glands that cause multiple small out pouchings. **Usually due to chronic reflux esophagitis**. There is controversy among the whole Candida situation. Per the Mayo GI book, Candida is often cultured but is not the causative factor.

Papilloma: The most common benign mucosal lesion of the esophagus. It's basically just hyperplastic squamous epithelium.

Feline Esophagus: Another Aunt Minnie. You have transient, fine transverse folds which course the esophagus. It can be normal. It can be associated with esophagitis..... *correlate clinically*.

Hernias: There are a bunch of ways to classify these, but the most common (and likely tested) is the relationship of the GE junction to the diaphragm. Axial (Sliding) types have the GE junction above the diaphragm. Paraesophageal (Rolling) types have the GE junction below the diaphragm, and a piece of the stomach above it. The rolling type has a higher rate of incarceration.

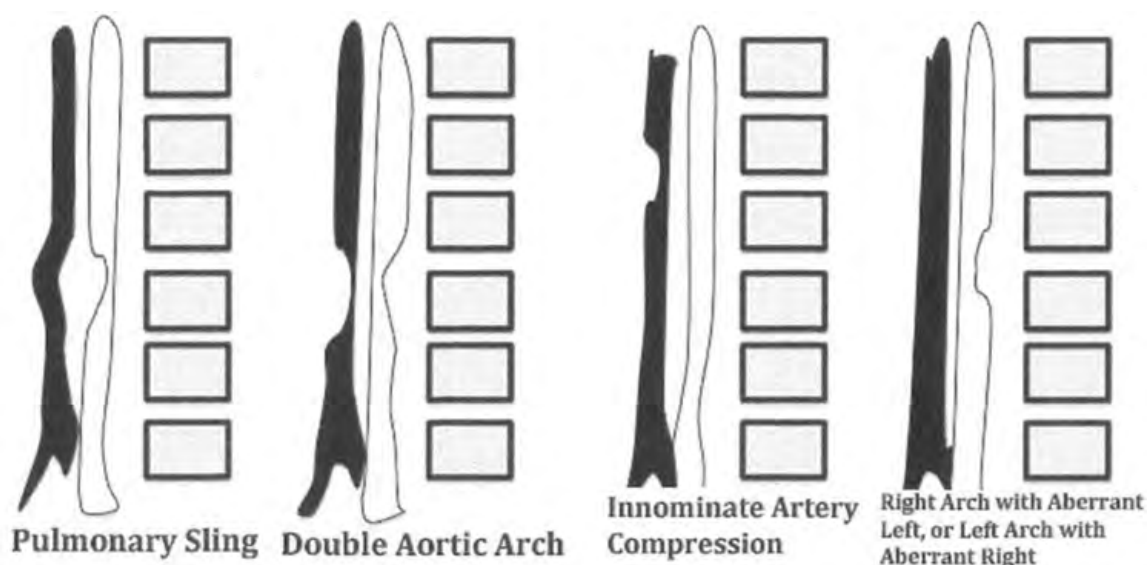
Esophageal Spasm: Basically tertiary contractions with pain. The term *nutcracker esophagus* requires manometric findings ($>180\text{mmHg}$).

Esophageal Web: Most commonly located at the cervical esophagus (near the cricopharyngeus). This thing is basically a ring caused by a thin mucosal membrane. There are 2 things to know about it:

- (1) It's a **risk factor for** esophageal and hypopharyngeal **carcinoma**
- (2) **Plummer -Vinson Syndrome:** iron def anemia, dysphagia, thyroid issues, "spoon-shaped nails"

Dysphagia Lusoria - THIS IS HIGH YIELD. Syndrome refers to problems swallowing secondary to compression from an aberrant right subclavian artery (most patients with aberrant rights don't have symptoms).

Vascular Impressions-THIS IS HIGH YIELD



Achalasia: A motor disorder where the distal 2/3 of the esophagus (smooth muscle part) doesn't have normal peristalsis ("absent primary peristalsis"), and the lower esophageal sphincter won't relax. Dilated esophagus above a smooth stricture at the GE junction (**Bird's Beak**). It's considered "**Vigorous Achalasia**" if it's an early / less severe form with the addition of repetitive simultaneous non-propulsive contractions. It's more common in women, but the secondary cancer occurs more in men.



Things to know:

- Failure of the Esophageal sphincter to relax
- Increased Risk of Candida
- Increased Risk of Cancer (Squamous Cell - 20 years later).

“Pseudoachalasia” (*secondary achalasia*) has the appearance of achalasia, but is secondary to a cancer at the GE junction. The difference is that real Achalasia will eventually relax, the pseudo won't.

Scleroderma: Affects the esophagus 80% of the time. Again the lower 2/3 of the esophagus stops working normally. The LES is incompetent and you end up with chronic reflux, which can cause scarring, Barrets, and even cancer (Adeno). They will show you lung changes (most commonly NSIP), and the barium esophagus (or a small bowel series showing closely spaced valvulae conniventes - hide bound).

Esophagus - High Yield Trivia

Path	Trivia
Esophagitis	Fold Thickening. May have smooth stricture at GE junction if severe
Barretts	Buzzword: Reticulated Mucosal Pattern
	Classically shown as Hiatal Hernia + High Stricture
Medication Induced Esophagitis	Ulcers; Usually at the level of the arch or distal esophagus
Crohns Esophagitis	Ulcers ; can be confluent in severe disease
Candidia	Discrete plaque like lesions that are seen as linear or irregular filling defects that tend to be longitudinally oriented, separated by normal mucosa
	Buzzword: Shaggy - when severe
	Not always from AIDS, can also be from motility disorders such as achalasia and scleroderma
Glycogen Acanthosis	Looks like Candidia, but in an asymptomatic old person
Herpes Ulcers	Multiple small, with Edema Halo (heipes has halo)
CMV / AIDS	Large Flat ulcers
Achalasia	Buzzword: Bird Beak, - smooth stricture at GE junction
	Path is failure of LES to relax (but it will slowly relax)
	Increased risk of Squamous Cell CA, and Candida
Pseudoachalsia	Cancer at the GE junction. Fixed Obstruction, will not relax
Scleroderma	Involves the Esophagus 80% of the time
	Looks a little like Achalasia (they will show you lung changes)
	Sequelae of reflux: stricture, barrets, cancer
Long Stricture	DDx: NG tube in too long, Radiation, Caustic Ingestion
Pseudodiverticulosis	Dilated submucosal glands, usually due to chronic reflux esophagitis.
	Esophageal stricture is seen in 90% of cases
Zenker Diverticula	Zenker in the back (above cricopharyngeus)
Killian-Jamieson	Lateral (below cricopharyngeus)

Stomach

Location Location:

- **H Pylori** Gastritis - Usually in **Antrum**
- **Zollinger-Ellison** - Ulcerations in the stomach (**jejunal ulcer** is the buzzword). Duodenal bulb is actually the most common location for ulcers in ZE.
- **Crohns** - Uncommon in the stomach, but when it is, it likes the **antrum**
- **Menetrier's** - Usually in the **Fundus** (*classically spares the antrum*)
- **Lymphoma** - **"Crosses the Pylorus"** - classically described as doing so, although in reality adenocarcinoma does it more.

Selective Polyposis Syndromes

Path	Trivia
Gardner Syndrome	FAP (Hyperplastic Stomach, Adenomatous Bowel Polyps) + Desmoid Tumors, Osteomas, Papillary Thyroid Cancer
Turcots	FAP (Hyperplastic Stomach, Adenomatous Bowel Polyps) + Gliomas and Medulloblastomas
Hereditary non polyposis Syndrome (Lynch)	DNA Mismatch Repair They get cancer everywhere in everything
Peutz-Jeghers	Hamartoma Style! Mucocutaneous Pigmentation
Cowden's	Small and Large Bowel CA + GYN CA Hamartoma Style! BREAST CA , Thyroid CA, Lhermitte-Dulcose (<i>posterior fossa noncancerous brain tumor</i>)
Cronkite-Canada	Hamartoma Style! Stomach, Small Bowel, Colon, Ectodermal Stuff (skin, hair, nails, yuck)
Juvenile Polyps	Hamartoma Style!
Zenker Diverticula	Zenker in the back (above cricopharyngeus)

THIS IS THE MOST LIKELY TO BE ASKED!!!!

GIST

- The most common mesenchymal tumor of the GI tract. (70% in stomach)
- Rare before age 40 (most in old people)
- A 90° angle is often formed between the edges of the mass and the normal gastric wall.
- They can be nasty, and met locally or distally. The sneaky thing (which would easily lend to a multiple choice question) is that **lymph node enlargement is NOT a classic feature**. Malignant GISTs tend to be large (>10cm)

Carneys Triad

“Carney’s Eat Garbage”

- Syndromes:

- o **Carney Triad:** Extra-Adrenal

- Pheochromocytoma, GIST, Pulmonary

- Chordoma (hamartoma)

- o NF-1

Chordoma

Extra Adrenal Pheo

GIST

HIGH YIELD: The Ridiculous Distinction Between Malignant Benign Ulcers with Barium

This vs That: Malignant vs Benign Ulcer (on Barium)

Malignant	Benign
Width > Depth	Depth > Width
Located within Lumen	Project behind the expected lumen
Nodular, Irregular Edges	Sharp Contour
Folds adjacent to ulcer	Folds radiate to ulcer
Aunt Minnie: Carmen Meniscus Sign	Aunt Minnie: Hampton’s Line

Gastric Cancer

It’s Either Lymphoma (<5%) or Carcinoma (95%).

Gastric carcinoma is usually a disease of an old person (median age 70). H. Pylori is the most tested risk factor. Ulcerated carcinoma (penetrating cancer) has the look of an advanced cancer 70% of the time. Metastatic spread to the ovary is referred to as a **Krukenberg Tumor**. Another high yield pearl: 2x - 6x- increased risk has been reported for development of carcinoma within the gastric remnant in patients with a gastroenterostomy performed for gastric ulcer disease (old school - prior to PPIs). Step 1 trivia question: swollen left supraclavicular node = **Virchow Node**.

Gastric Lymphoma, can be primary (MALT), or secondary to systemic lymphoma. The stomach is the most common extranodal site for non-Hodgkin lymphoma. Even when extensive, it **rarely causes gastric outlet obstruction**. It was classically described as "*crossing the pylorus*", although since gastric carcinoma is like 10x more common it is actually more likely to do that. Has multiple looks and can be big, little, ulcerative, polypoid, or look like target lesions. It can also look like Linitis Plastica. *It can rupture with treatment (chemo)*.

Gastric Cancer More Likely

- *More Likely to Cause Gastric Outlet Obstruction*
- *More Likely to be in the distal stomach*
- *More Likely to extend beyond the serosa and obliterate adjacent fat plains*
- *More Likely to be a focal mass (95% of primary gastric tumors are adenocarcinoma)*

Linitis Plastica: The leather bottle stomach. It's the result of a scirrhous adenocarcinoma, with diffuse infiltration. Can be from **breast** or lung mets.

Misc. Gastric Conditions

Menetrier's Disease: Rare and has a French sounding name, so it's almost guaranteed to be on the test. It's an idiopathic gastropathy. Rugal thickening classically involves the fundus and spares the antrum. Bimodal age distribution (childhood form thought to be CMV related). They end up with low albumin, from loss into gastric lumen.

Ram's Horn Deformity (*Pseudo Billroth 1*) Tapering of the antrum, is said to look like a Ram's Horn. This is a differential case, and can be seen with Scarring via peptic ulcers, Granulomatous Disease (Crohns, Sarcoid, TB, Syphilis), or Scirrhous Carcinoma. **The stomach is the most common location for sarcoid (in the GI tract).**

Gastric Volvulus

Two Flavors:

- *Organoaxial* - the greater curvature flips over the lesser curvature. This is seen in old ladies with paraesophageal hernias. It's way more common.
- *Mesenteroaxial* - twisting over the mesentery. Can cause ischemia and needs to be fixed. Additionally this type causes obstruction. This type is more common in kids.

Gastric Diverticulum: The way they always ask this, is by trying to get you to call it an adrenal mass (it's most commonly in the posterior fundus). Find the normal adrenal.

Gastric Varices: This gets mentioned in the pancreas section, but **I** just want to hammer home that test writers love to ask splenic vein thrombus causing isolated gastric varices. Some sneaky ways they can ask this is by saying "pancreatic cancer" or "Pancreatitis" causes gastric varices. Which is true.... because they are associated with splenic vein thrombus. So, just watch out for that.

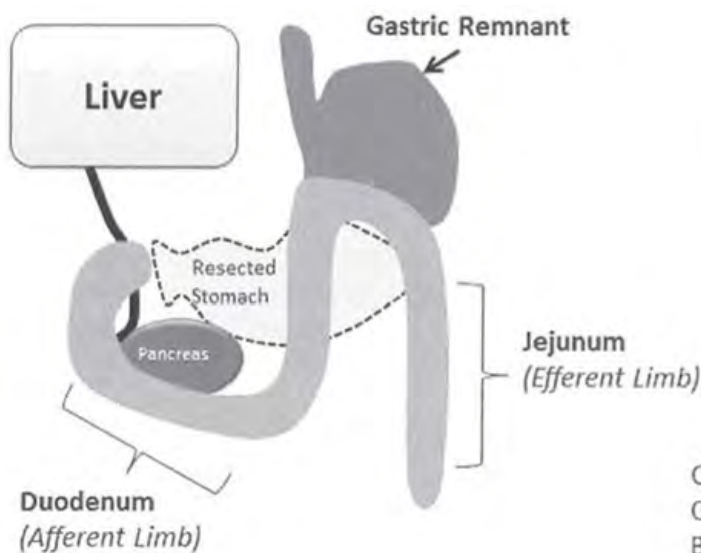
Areae Gastricae: This is a **normal** fine reticular pattern seen on double contrast. Multiple choice writers have been known to ask when does this "enlarge"? The answer is that it enlarges in elderly and patient's with H. Pylori. Also it can focally enlarge next to an ulcer. It becomes obliterated by cancer or atrophic gastritis.

Chronic Aspirin Therapy: "Multiple gastric ulcers" is the buzzword. Obviously this is non-specific, but some sources say it occurs in 80% of patient's with chronic aspirin use. As a point of trivia, aspirin does NOT cause duodenal ulcers. If you see multiple duodenal ulcers (most duodenal ulcers are solitary) you should think Zollinger-Ellison.

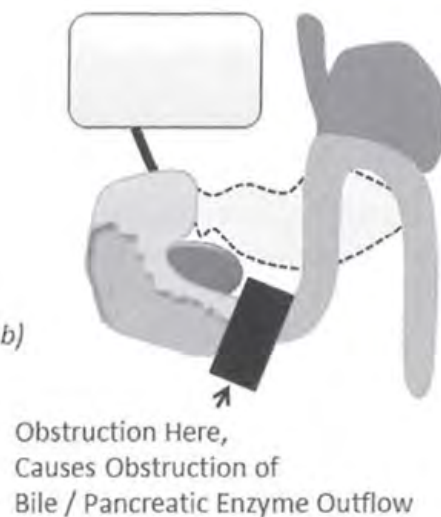
Upper GI Surgical Complications:

Afferent Loop Syndrome: An uncommon complication post billroth 2. The most common cause is obstruction (adhesions tumor, intestinal hernia) of the afferent. The acute form may have a closed loop obstruction. The result of this afferent obstruction is the build up of biliary, pancreatic, and intestinal secretions resulting in afferent limb dilation. The back pressure from all this back up dilates the gallbladder, and causes pancreatitis. A much less common cause is if the stomach preferentially drains into the afferent loop.

Billroth 2



Afferent Limb Syndrome



Jejunogastric Intussusception: This is a rare complication of gastroenterostomy. The Jejunum herniates back into the stomach (usually the efferent limb) and can cause gastric obstruction. High mortality is present with the acute form.

Bile Reflex Gastritis: Fold thickening and filling defects seen in the stomach after Billroth I or II are likely the result of bile acid reflux.

Gastro-Gastric Fistula: This is seen in Roux-en-Y patients who gain weight years later. The anastomotic breakdown is a chronic process, and often is not painful.

Cancer: With regard to these old peptic ulcer surgeries (billroths), there is a 3-6 times increased risk of getting adenocarcinoma in the gastric remnant (like 15 years after the surgery).

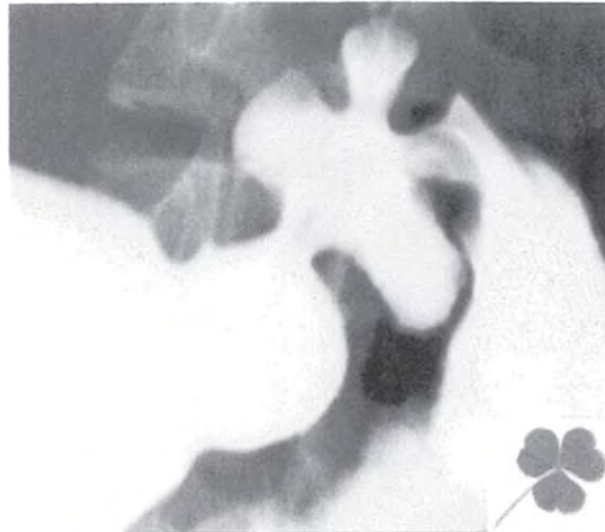
Small Bowel

The Target Sign:

'Single Target: GIST, Primary Adenocarcinoma, Lymphoma, Ectopic Pancreatic Rest, Met (Melanoma).

'Multiple Target: Lymphoma, Met (Melanoma)

Clover Leaf Sign: This is an Aunt Minnie for Healed Peptic Ulcer of the Duodenal Bulb.



Clover Leaf Sign - Healed Duodenal Ulcer

Bowel Folds:

Let's be honest Fluoro is pretty useless compared to endoscopy and CT. But just for fun let's pretend it's 1950.

One search pattern to organize your thinking on the subject:

- (1) Folds
- (2) Filling Defects
- (3) Loop Separation

With regard to fold thickening, the best way to think about this is categorically

- Thin Straight Folds with a dilated lumen
- Thick (>3mm) Straight folds - of which they can be diffuse or segmental, and
- Thick Nodular folds - of which they can be diffuse or segmental.

Each subtype will carry a different differential.

j Fold Pattern				
Thin Folds, with Dilation	Thick Folds > 3mm		Thick Folds with Nodularity	
	Segmental	Diffuse	Segmental	Diffuse
Mechanical Obstruction	Ischemia	Low Protein	Crohns	Whipples
Paralytic Ileus	Radiation	Venous Congestion	Infection	Lymphoid Hyperplasia
Scleroderma	Hemorrhage	Cirrhosis	Lymphoma	Lymphoma
Sprue	Adjacent Inflammation		Mets	Mets
				Intestinal Lymphangiectasia

With regard to filling defects, the way to think about this is:

- Uniform 2-4mm nodules = Lymphoid Hyperplasia
- Nodules of larger or varying sizes = Cancer (probably mets and therefore probably melanoma).

Loop Separation can be thought about as with or without tethering

- Without Tethering = Ascites, Wall Thickening (Crohns, Lymphoma), Adenopathy, or Mesenteric Tumors
- With Tethering = Just say carcinoid
- A pearl is that extrinsic processes will spare the mucosa, intrinsic process will alter the mucosa.

Selected Small Bowel Path:

Whipples: Rare infection (Tropheryma Whipplei)

Likes white men in their 50s. The bug infiltrates the lamina propria with large macrophages infected by intracellular whipple bacilli leading to marked swelling of intestinal villi and thickened irregular mucosal folds primarily in duodenum and proximal jejunum. The buzzword is “sand like nodules” referring to diffuse micronodules in jejunum. Jejunal mucosal folds are thickened. This is another cause of low density (near fat) enlarged lymph nodes.

Pseudo Whipples: MAI infection. Seen in AIDS patients with CD4<100. Nodules in the jejunum, just like regular Whipples is the finding (plus a big spleen, and retroperitoneal lymph nodes).

Celiac Sprue: Small bowel malabsorption of gluten.

- High yield points:
 - o Can cause malabsorption of iron, and lead to iron deficiency anemia,
 - o Associated with Idiopathic Pulmonary Hemosiderosis (Lane Hamilton Syndrome)
 - o Increased Risk of bowel wall **lymphoma**
 - o Gold standard is biopsy (*surprisingly not barium*)
 - o Dermatitis Herpetiformis - some skin thing (remember that from step 1)
- Findings (CT / Barium)
 - o **Fold Reversal** is the Buzzword (Jejunum like Ileum, Ileum like Jejunum)
 - o Moulage Sign - dilated bowel with effaced folds (tube with wax poured in it)
 - o **Cavitary Lymph Nodes (low density)**
 - o Splenic Atrophy

Intestinal Lymphangiectasia: Lymphangiectasia results from obstruction to the flow of lymph from the small intestine into the mesentery. This results in dilation of the intestinal and serosal lymphatic channels. This can be primary from lymphatic hypoplasia, or secondary from obstruction of the thoracic duct (or any place in between).

SMA Syndrome: This is an obstruction of the 3rd portion of the duodenum by the SMA (it pinches the duodenum in the midline). It is seen in **patients who have recently lost a lot of weight**.

Graft vs Host: Buzzword = **Ribbon bowel**. It occurs in patients after bone marrow transplant. It's less common with modern anti-rejection drugs. Skin, Liver, and GI tract get hit. Small bowel is usually the most severely affected. Bowel is featureless, atrophic, and has fold thickening (ribbon like).

Meckel's Diverticulum / Diverticulitis: This is a congenital true diverticulum of the distal ileum. A piece of total trivia is that it is a persistent piece of the omphalomesenteric duct. Step 1 style, "rule of 2s" occurs in 2% of the population, has 2 types of heterotopic mucosa (gastric and pancreatic), located 2 feet from the IC valve, it's usually 2 inches long (and 2cm in diameter), and usually has symptoms before the child is 2. If it has gastric mucosa (the ones that bleed typically do) it will take up Tc-Pertechnetate just like the stomach (hence the Meckel's scan).

High Yield Meckel's Trivia (Regarding Complications)

- Can get diverticulitis in the Meckels (mimic appendix)
- GI Bleed from Gastric Mucosa (causes 30% of symptomatic cases)
- Can be a lead point for intussusception (seen with inverted diverticulum)
- Can cause Obstruction

Barium Bowel Buzzwords

Infections that like the Duodenum
(and proximal small bowel)

Giardia

Strongyloides

Infections that like the Terminal Ileum

TB

Yersinia

Fold Reversal

Ribbon Bowel

Lead Pipe

String Sign

Hide Bound

Coned Shaped

Cecum

Celiac Sprue

Graft vs Host

Ulcerative Colitis

Crohns

Scleroderma

Amebiasis

Duodenal Inflammatory Disease: You can have fold thickening of the duodenum from adjacent inflammatory processes of the pancreas or gallbladder. You can also have thickening and fistula formation with Crohn's (usually when the colon is the primary site). Primary duodenal Crohns can happen, but is super rare. **Chronic dialysis patients may get severely thickened duodenal folds** which can mimic the appearance of pancreatitis on barium.

Jejunal Diverticulosis: Less common than colonic diverticulosis, but does occur. They occur along the mesenteric border. Important **association is bacterial overgrowth and malabsorption**. They could show this with CT, but more likely will show it with barium (if they show it at all).

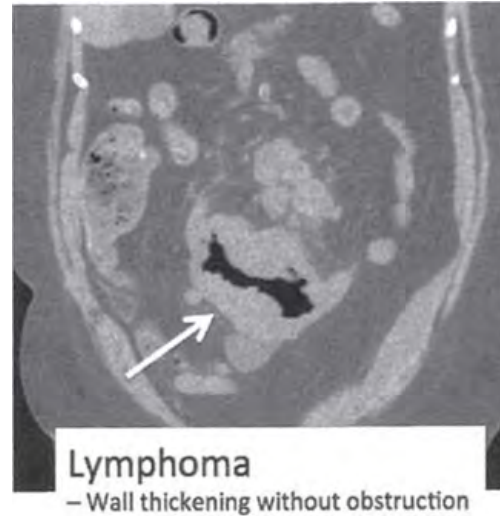
Small Bowel Cancer

Adenocarcinoma: Most common in the proximal small bowel (usually duodenum).

Increased incidence with celiac disease and regional enteritis. Focal circumferential bowel wall thickening in proximal small bowel is characteristic on CT.

Lymphoma: It's usually the non-Hodgkin flavor. Patient's with celiac, Crohns, AIDS, and SLE are higher risk. It can look like anything (infiltrative, polypoid, multiple nodules etc....). **Key piece of trivia is they usually do NOT obstruct, even with massive circumferential involvement.**

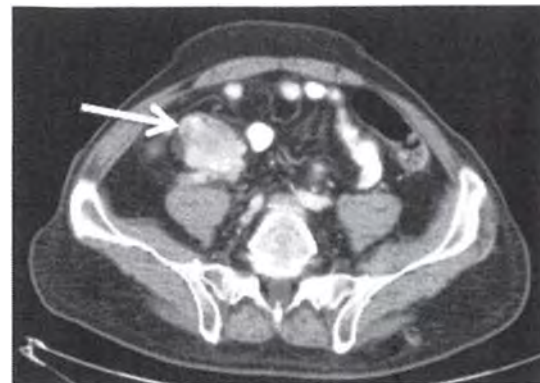
The Hodgkin subtype is more likely to cause a desmoplastic reaction.



Carcinoid: This has an **Aunt Minnie look with a mass + desmoplastic stranding.**

“Starburst” appearance of the mesenteric mass with calcifications. This tumor most commonly occurs in young adults. The primary tumor is often not seen. Liver mets are often hypervascular. Step 1 style, you don't get carcinoid syndrome (flushing, diarrhea) until you met to the liver. The **most common primary location is the distal appendix (50%).** The appendix + terminal ileum makes it 90%. The

appendix, has the best prognosis of all G1 primary sites. Systemic serotonin degrades the heart valves (right sided), and classically causes tricuspid regurgitation - *more on this in the cardiac chapter.* MIBG or Octreotide scans can assist with diagnosis and staging - *more on this in the nuclear medicine chapter.*



Mets: This is usually **melanoma** (which hits the small bowel 50% in fatal cases). You can also get hematogenous seeding of the small bowel with breast, lung, and Kaposi sarcoma. Melanoma will classically have multiple targets.

Hernias:

Inguinal Hernias: - the most common type of abdominal wall hernia. M>F (7:1)

Direct	Indirect
Less common	More Common
Medial to inferior epigastric artery	Lateral to inferior epigastric artery
Defect in Hesselback Triangle	Failure of processus vaginalis to close
NOT covered by internal spermatic fascia	Covered by internal spermatic fascia

Femoral: Likely to obstruct, and seen in old ladies. They are medial to the femoral vein, and posterior to the inguinal ligament (usually on the right).

Obturator Hernia: Another old lady hernia. Often seen in patients with increased intraabdominal pressure (Ascites, COPD - chronic cough). Usually asymptomatic - but can strangulate.

Lumbar Hernia: Can be superior (Grynfeltt-Lesshaft) through the superior lumbar triangle, or inferior (Petit) through the inferior lumbar triangle. Superior is more common than inferior. Otherwise, they are very similar and usually discussed together. Causes are congenital or acquired (post-surgery or acquired).

Spigelian Hernia: The question is probably the location along the Semilunar line (“S” for “S”) through the transversus abdominus aponeurosis close to the level of the arcuate line.

Richter Hernia: Contains only one wall of bowel and therefore does not obstruct.

Littre Hernia : Hernia with a Meckel Diverticulum in it.

Amyand Hernia: Hernia with the appendix in it.

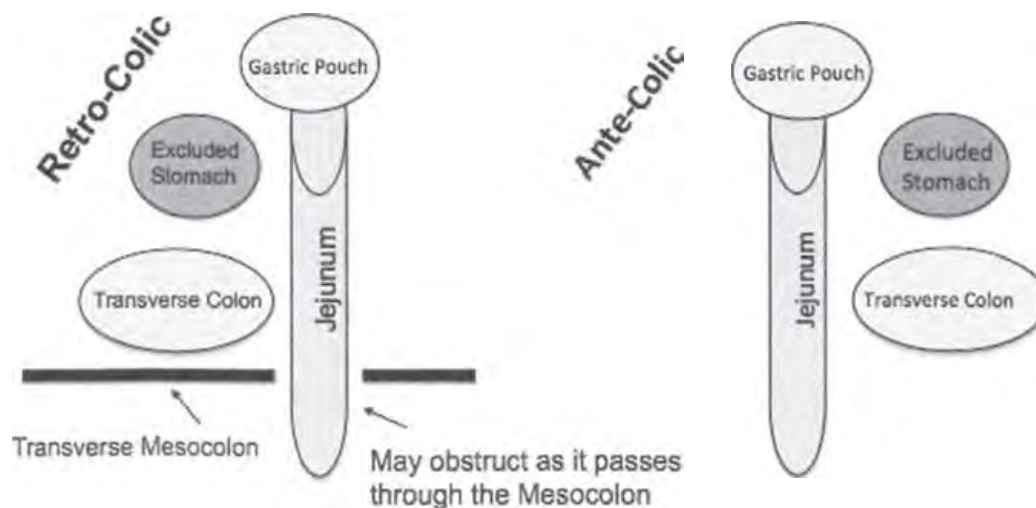
Hernias Post Laparoscopic Roux-en-Y Gastric Bypass:

Factors that promote internal hernia by bypass: (1) Laparoscopic over Open - supposedly creates less adhesion, so you have more mobility (2) Degree of weight loss ; more weight loss = less protective, space occupying mesenteric fat.

There are 3 potential sites.

- (1) At the defect in the transverse mesocolon, through which the Roux-Loop Passes (if it's done in the retrocolic position).
- (2) At the mesenteric defect at the enteroenterostomy
- (3) Behind the Roux limb mesentery placed in a retrocolic or antecolic position
(**retrocolic Petersen** and **antecolic Petersen** type). ** This is the one they will likely ask because it has an eponym with it.

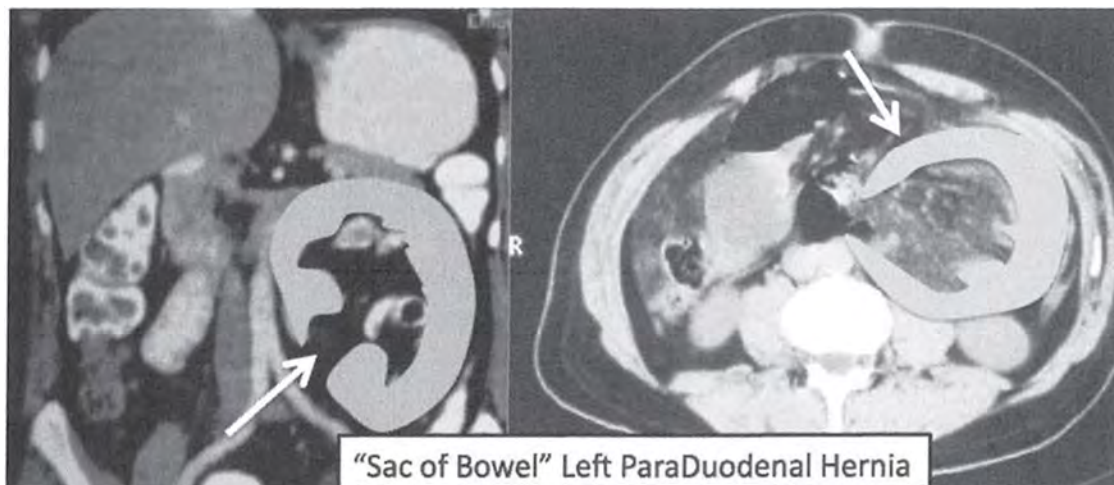
Retro-Colic vs Ante-Colic Surgical Methods



Internal Hernia: These can be sneaky. The most common manifestation is closed loop obstruction (often with strangulation). There are 9 different subtypes, of which I refuse to cover. I will touch on the most common, and the general concept.

General Concept: This is a herniation of viscera, through the peritoneum or mesentery. The herniation takes place through a known anatomic foramina or recess, or one that has been created post operatively.

Paraduodenal (right or left): This is by far the most common type of internal hernia. They can occur in 5 different areas, but it's much simpler to think of them as left or right. Actually, **75% of the time they are on the left.** The exact location is the duodenojejunal junction ("fossa of Landzert"). Here is the trick, the herniated small bowel can become trapped in a **"sac of bowel,"** between the pancreas and stomach to the left of the ligament of Treitz. The sac characteristically contains the I MV and the left colic artery.



The right sided PDHs are located just behind the SMA and just below the transverse segment of the duodenum, at the "Fossa of Waldeyer." The classic setting for right sided PDHs is non-rotated small bowel, with normally rotated large bowel.

Mesenteric Ischemia: *This will be discussed in the vascular chapter.*

Large Bowel

Crohns Disease vs Ulcerative Colitis:

Crohns Disease: Typically seen in a young adult (15-30), but has a second smaller peak 60-70. Discontinuous involvement of the entire GI tract (mouth -> asshole). Stomach, usually involves antrum (Ram's Horn Deformity). Duodenal involvement is rare, and NEVER occurs without antrum involvement. Small bowel is involved 80% of the time, with the terminal ileum almost always involved (Marked Narrowing = String Sign). After surgery the "neo-terminal ileum" will frequently be involved. The colon involvement is usually right sided, and often spares the rectum / sigmoid. Complications include fistulae, abscess, gallstones, fatty liver, and sacroiliitis.

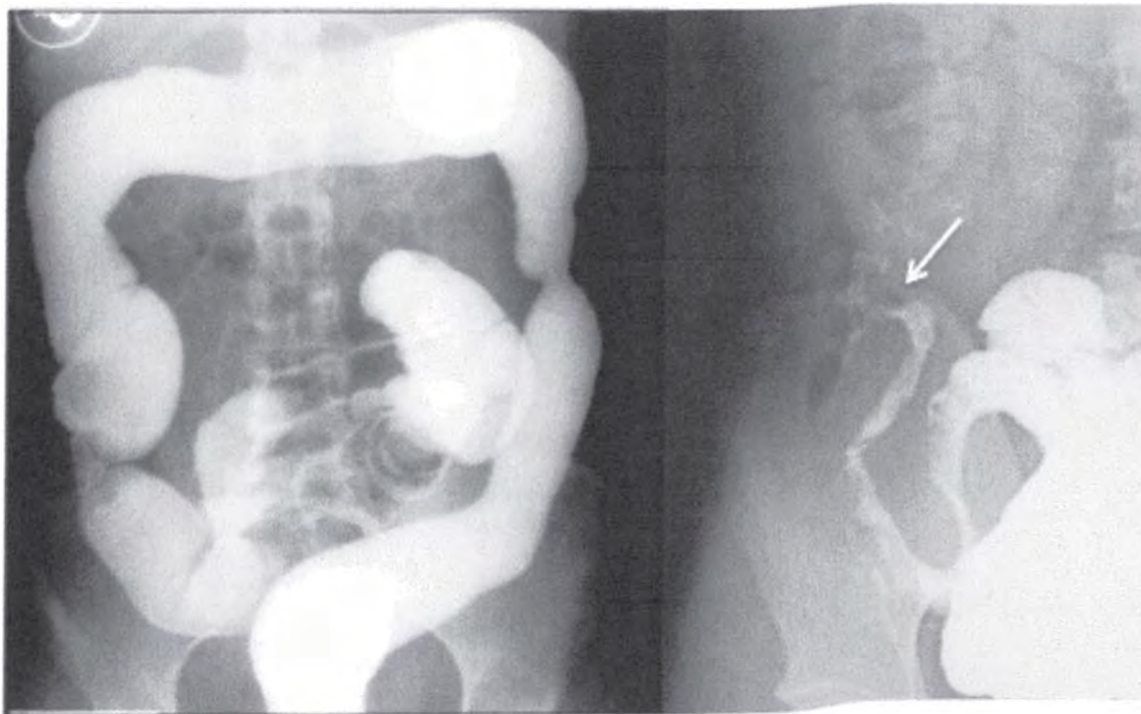
Crohns Buzzwords

Squaring of the folds	An early manifestation from obstructive lymphedema
Skip lesions	Discontinuous involvement of the bowel
Proud loops	Separation of the loops caused by infiltration of the mesentery, increase in mesenteric fat and enlarged lymph nodes
Cobblestoning	Irregular, appearance to bowel wall caused by longitudinal and transverse ulcers separated by areas of edema
Pseudopolyps	Islands of hyperplastic mucosa
Filiform	Post-inflammatory polyps - long and worm like
Pseudodiverticula	Found on anti-mesenteric side. From bulging area of normal wall opposite side of scarring from disease,
String-sign	Marked narrowing of terminal ileum from a combination of edema, spasm and fibrosis;

Ulcerative Colitis: Just like Crohns, it typically occurs in a “young adult” (age 15-40), with a second peak at 60-70. Favors the male gender. It **involves the rectum 95% of the time, and has retrograde progression**. Terminal ileum is involved 5-10% of the time via backwash ileitis (wide open appearance). It is continuous and does not “skip” like Crohns. It is associated with Colon Cancer, Primary Sclerosing Cholangitis, and Arthritis (similar to Ankylosing Spondylitis). On Barium, it is said that the colon is ahastral, with a diffuse granular appearing mucosa. “Lead Pipe” is the buzzword (shortened from fibrosis). Here is a key clinical point; UC has an increased risk of cancer (probably higher than Crohns), and it doesn’t classically have enlarged lymph nodes (like Crohns does), so if you see a big lymph node in a UC patient (especially one with long standing disease) you have to think that might be cancer.

More Common In : Crohns vs UC

Path	More Common IN
Gallstones	Crohns
Primary Sclerosing Cholangitis	Ulcerative Coliits
Hepatic Abscess	Crohns
Pancreatitis	Crohns



Ulcerative Colitis

- Haustral Loss, Lead Pipe Appearance

Crohns

- String Sign at IC Valve

Crohns vs Ulcerative Colitis

Crohns	UC
Slightly less common in the USA	Slightly more common in the USA
Discontinuous “Skips”	Continuous
Terminal Ileum - <i>String Sign</i>	Rectum
Ileocecal Valve “Stenosed”	Ileocecal Valve “Open”
Mesenteric Fat Increased “ <i>creepingfat</i> ”	Perirectal fat Increased
Lymph nodes are usually enlarged	Lymph nodes are NOT usually enlarged
Makes Fistula	Doesn’t Usually Make Fistula

Diverticulosis / Diverticulitis: Some trivia worth knowing is that diverticulosis actually bleeds more than diverticulitis. Right sided is less common (but is seen in young Asians). Fistula formation is actually most common with diverticulitis, and can occur to anything around it (another piece of bowel, the bladder, etc..).

Epiploic Appendagitis / Omental Infarct: Epiploic appendages along the serosal surface of the colon can torse, **most commonly on the left**. There is not typically concentric bowel wall thickening (unlike diverticulitis). *Omental infarction* is typically a larger mass with a more oval shape, and central low density. It is **more commonly on the right** (*ROI-right omental infarct*). Both entities are self limiting.

Infections:

Entamoeba Histolytica: Parasite that causes bloody diarrhea. Can cause liver abscess, spleen abscess, or even brain abscess. Within the colon it is one of the causes of toxic megacolon. They are typically “flask shaped ulcers” on endoscopy. With regard to barium, the buzzword is “*coned cecum*” referring to a change in the normal bulbous appearance of the cecum, to that of a cone. It affects the cecum and ascending colon most commonly, and unlike many other GI infections **sparing the terminal ileum**.

Colonic TB: Typically involves the terminal ileum, and is another cause of the “**coned cecum**” appearance. Causes both ulcers and areas of narrowing. Two other signs: (1) *Fleischner sign* - enlarged gaping IC valve, and (2) *Stierlin sign* - narrowing of the TI.

Colonic CMV: Seen in patients who are immunosuppressed. Causes deep ulcerations - which can lead to perforation. Step 1 question = Cowdry Type A intranuclear inclusion bodies

C-Diff: Classically seen after antibiotic therapy, the toxin leads to a super high WBC count. CT findings of the “*accordion sign*” with contrast trapped inside mucosal folds is always described in review books and is fair game for multiple choice. The barium findings include thumbprinting, ulceration, and irregularity. Of course it can cause toxic megacolon as mentioned above.

Neutropenic Colitis (Typhlitis): Infection **limited to the cecum** occurring in severe neutropenia.

Other Random High Yield Conditions:

Adenocarcinoma: Common cause of cancer death (#2 overall). The cancer on the right tends to bleed (present with blood stools, anemia), the cancer on the left tends to obstruct. Apple core is a buzzword (*if you didn't know that by now close this book, quit medicine, and apply to dental school*).



Apple Core Lesion - Cancer

Squamous Carcinoma - Occasionally arises in the anus (*think HPV*).

Adenoma - The most common benign tumor of the colon and rectum. The *villous adenoma* has the largest risk for malignancy.

Rectal Cancer

Things to know:

- Nearly always (98%) adenocarcinoma
- If the path says Squamous - the cause was HPV (*use your imagination on how it got there*).
- Total mesorectal excision is standard surgical method
- Lower rectal cancer (0-5 cm from the anorectal angle), has the highest recurrence rate.
- MRI is used to stage.
- Stage T3 - called when tumor breaks out of the rectum and into the perirectal fat.

Appendicitis - The classic pathways are: obstruction (fecalith or reactive lymphoid tissue) -> mucinous fluid builds up increasing pressure -> venous supply is compressed -> necrosis starts -> wall breaks down -> bacteria get into wall -> inflammation causes vague pain (umbilicus) -> inflamed appendix gets larger and touches parietal peritoneum (pain shifts to RLQ). It occurs in an adolescent or young adult (or any age). The measurement of 6mm, was originally described with data from ultrasound compression, but people still generally use it for CT as well. Secondary signs of inflammation are probably more reliable for CT.

Appendix Mucocele - Mucinous cystadenomas are the most common mucinous tumor of the appendix. They produce mucin and can really dilate up and get big. They look similar to cystadenocarcinomas and can perforate leading to pseudomyxoma peritonei. On ultrasound the presence of an “**onion sign**” - layering within cystic mass is a suggestive feature of a mucocele.

Lipomas: The second most common tumor in the colon.

Colonic Volvulus: Comes in several flavors:

Sigmoid: Most common adult form. Seen in the nursing home patient (chronic constipation is a predisposing factor). Buzzword is coffee bean sign (or inverted 3 sign). Another less common buzzword is Frimann Dahl’s sign - which refers to 3 dense lines converging towards the site of obstruction. Points to the RUQ. Recurrence rate after decompression = 50%.

Cecal: Seen in a younger person (20-40). Associated with people with a “long mesentery.” More often points to the LUQ. Much less common than sigmoid.

Cecal Bascule: Anterior folding of the cecum, **without twisting**. A lot of surgical text books dispute this thing even being real (they think it’s a focal ileus). The finding is supposedly dilation of the cecum in an ectopic position in the middle abdomen, without a mesenteric twist.

This vs That: **Sigmoid Volvulus vs Cecal Volvulus**

Sigmoid	Cecal
Old Person	Younger Person
Points to the RUQ	Points to the LUQ

Toxic Megacolon: Ulcerative colitis, and to a lesser degree Crohns is the primary cause. C-Diff can also cause it. Gaseous dilation distends the transverse colon (on upright films), and the right and left colon on supine films. **Lack of haustra** and pseudopolyps are also seen. Some people say the presence of normal haustra excludes the diagnosis. **Don’t do a barium enema** because of the risk of perforation. Another piece of trivia is that peritonitis can occur without perforation.

Behcets: Ulcers of the penis and mouth. Can also affect GI tract (and **looks like Crohns**) - *most commonly affects the ileocecal region*. It is also cause of **pulmonary artery aneurysms** (test writers like to ask that).

Colonic Pseudo-Obstruction (*Colonic Ileus, Ogilvie Syndrome*): Usually seen after serious medical conditions, and nursing home patients. It can persist for years, or progress to bowel necrosis and perforation. The classic look is marked diffuse dilation of the large bowel, without a discrete transition point.

Diversion Colitis: Bacterial overgrowth in a blind loop through which stool does not pass (any surgery that does this).

Colitis Cystica: This cystic dilation of the mucous glands comes in two flavors: Superficial or Profunda (Deep).

Superficial: The superficial kind consists of cysts that are small in the entire colon. It's associated with vitamin deficiencies, and tropical sprue. Can also be seen in terminal leukemia, uremia, thyroid toxicosis, and mercury poisoning.

Profunda: These cysts may be large and are seen in the pelvic colon and rectum.



Rectal Cavernous Hemangioma: Obviously very rare. Just know it's associated with a few syndromes; Klippel-Trenaunay-Weber, and Blue Rubber Bleb. They might show you a ton of phleboliths down there.

Gossypiboma: This isn't really a GI pathology but it's an abscess mimic. It's a retained cotton product or surgical sponge, and can elicit an inflammatory response.

Barium Gone Bad

Complications of barium use are rare, but can be very serious. They come in two main flavors: (1) Peritonitis, and (2) Intravasation.

Barium Peritonitis: This is why you use a water soluble contrast any time you are worried about leak. The pathology is an attack of the peritoneal barium by the leukocytes which creates a massive inflammatory reaction (often with massive ascites and sometimes hypovolemia and resulting shock). In the event of this complication, you should give IV fluids to reduce the risk of hypovolemic shock. The long term sequela of barium peritonitis is the development of granulomas and adhesions (causing obstructions etc..).

Barium Intravasation: This is super rare, but can happen. If barium ends up in the systemic circulation it results in death about 50% of the time (often immediate - from pulmonary embolism). Risk is increased with inflammatory bowel or diverticulitis (altered mucosa).

Peritoneal Cavity

Pseudomyxoma Peritonei: This is a gelatinous ascites that results from either (a) ruptured mucocoele (usually appendix), or interperitoneal spread of a mucinous neoplasm (ovary, colon, appendix, and pancreas). It's usually the appendix (least common is the pancreas). The buzzword is "**scalloped appearance of the liver.**" Recurrent bowel obstructions are common.

Peritoneal Carcinomatosis: The main thing to know regarding peritoneal implants is that the natural flow of ascites dictates the location of implants. This is why the **retrovesical space is the most common spot**, since it's the most dependent part of the peritoneal cavity.

Omental Seeding/Caking: The omental surface can get implanted by cancer, and become thick (like a mass). The catch-phrase is "posterior displacement of the bowel - from the anterior abdominal wall."

Primary Peritoneal Mesothelioma: This is super rare. People think about mesothelioma involving the pleura (and it does 75% of the time). The other 25% of the time it involves the peritoneal surface. The thing to know is that it occurs 30-40 years after the initial asbestosis exposure

Cystic Peritoneal Mesothelioma: This is the even more rare benign mesothelioma, that is NOT associated with prior asbestos exposure. It usually involves a women of child bearing age (30s).

Mesenteric Lymphoma: This is usually non-Hodgkin lymphoma, which supposedly involves the mesentery 50% of the time. The **buzzword is "sandwich sign."** The typical appearance is a lobulated confluent soft tissue mass encasing the mesenteric vessels "sandwiching them."

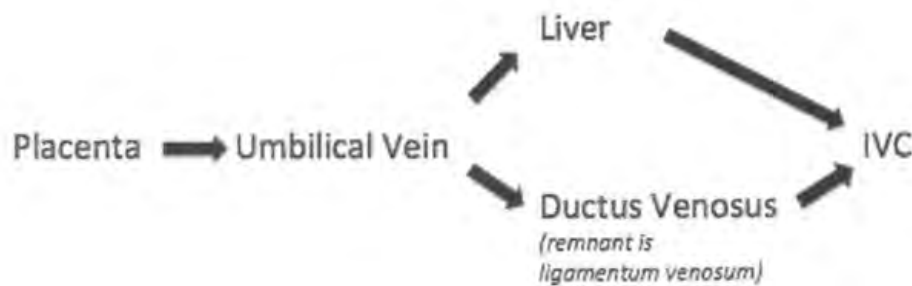
The Liver and Biliary System

Anatomy Trivia

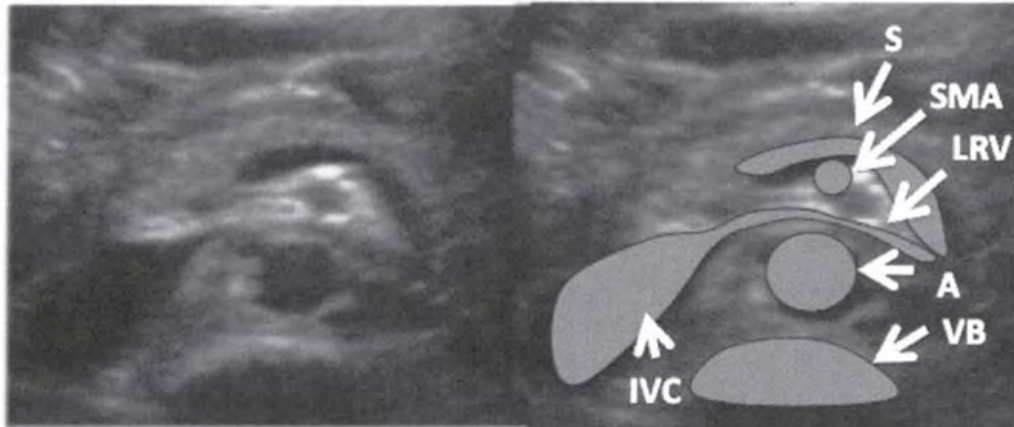
The liver is covered by visceral peritoneum except at the porta hepatis, bare area, and the gallbladder fossa. An injury to the “bare area” can result in a retroperitoneal bleed. Functional division of the liver into multiple segments is done by the Couinaud system. The caudate lobe (segment 1) has a direct connection to the IVC through its own hepatic veins, which do not communicate with the primary hepatic veins. Additionally, the caudate is supplied by branches of both the right and left portal veins - which matters because the caudate may be spared or hypertrophy as the result of various pathologies, Budd Chiari, etc... (as discussed below). Along the same lines of anatomy explaining pathology, the intra-hepatic course of the right portal vein is longer than the left, which is why it is more susceptible to fibrosis (*this is why the right liver shrinks, and the left liver grows in cirrhotic morphology*). The replaced right hepatic (origin from the SMA) is the most common anatomic variant.

Normal MRI Signal Characteristics: I like to think of the spleen as a bag of water/blood (T2 bright, T1 dark). The pancreas is the “brightest T1 structure in the body” because it has enzymes. Well, the liver also has enzymes and is similar to the pancreas (T1 Brighter, T2 darker).

Fetal Circulation: The fetal circulation anatomy is high yield anatomic trivia.



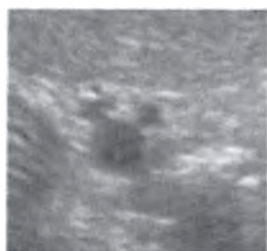
Classic Ultrasound Anatomy: There are 5 high yield looks, that are classically tested with regard to ultrasound anatomy. In years past, these were said to have been shown by oral boards examiners (likely the same dinosaurs writing the CORE exam).



S = Splenic Vein, SMA = SMA, LRV = Left Renal Vein,
A= Aorta, VB = Vertebra Body, IVC = IVC



RHA - Right Hepatic Artery, CBD = Common Bile Duct,
PV = Portal Vein



Mickey Mouse Sign: (1) Bile Duct, (2)
Hepatic Artery, (3) Portal Vein



Fat in the Falciform Ligament /
Ligamentum Teres

Blah Blah Blah

“Hey! Let’s talk about the liver, cirrhosis, portal HTN, and the development of HCC”

My idea is that by leading with a discussion of normal physiology, and the changes that occur with diffuse liver injury, that a lot of the processes and changes that occur with cirrhosis will make more sense (and be easier to remember). If you are in a rush to cram for the test just skip this discussion and move on to the charts. If you have more time, I think understanding the physiology is worthwhile.

Hepatocyte injury can occur from a variety of causes including viruses, alcohol, toxins (*alfatoxins i.e. peanut fungus*), and nonalcoholic fatty liver disease. These injuries result in increased liver cell turnover, to which the body reacts by forming regenerative nodules. The formation of regenerative nodules is an attempt by the liver not just to replace the damaged hepatocytes but also to compensate for lost liver function. In addition to activation of hepatocytes, stellate cells living in the space of Disse become active and proliferate changing into a *myofibroblast-like cell* that produces collagen. This collagen deposition causes fibrosis.

The development of fibrosis first puts the squeeze on the right portal vein (which *usually* has a longer intrahepatic course). This causes atrophy of segments 6 and 7, and compensatory hypertrophy of the caudate, segments 2, and 3. Because these changes, some people will try and use a caudate / right lobe ratio ($C/RL > 0.75$ is 99% *specific*), to call cirrhosis.

All this squeezing can lead to portal hypertension. Portal hypertension is usually the result of increased hepatic resistance from pre-hepatic (*portal vein thrombosis, tumor compression*), hepatic (*cirrhosis, schistosomiasis*) and post hepatic (*Budd-Chiari*) causes. Obviously, most cases are hepatic with schistosomiasis being the most common cause world-wide, and EtOH cirrhosis being the most common cause in the US. Once portal venous pressure exceeds hepatic venous pressure by 8 mmHg - portal hypertension has occurred. In reaction to this increased resistance offered by the liver, collaterals will form to decompress the liver by carrying blood away from it. These tend to be esophageal and gastric varices. As a point of trivia, in pre-hepatic portal hypertension, collaterals will form above the diaphragm and in the hepatogastric ligaments to bypass the obstruction.

The liver has a dual blood supply (70% portal, 30% hepatic artery) with compensatory relationships between the two inflows; arterial flow increases as portal flow decreases. This helps explain the relationship between these two vessels with regard to Doppler US. As fibrosis leads to portal hypertension, velocity in the hepatic artery increases.

You will sometimes see perfusion related changes in the setting of cirrhosis. Since the fibrosis blockade takes place at the level of the central lobular vein (into sinusoids), flow remains adequate for the central zones of the liver, but not for the peripheral zones. The arterial response produces enhancement of the peripheral subcapsular hepatic parenchyma with relative hypodensity of the central perihilar area. The consequent CT pattern is referred to as the “central-peripheral” phenomenon.

Sometimes you will see reversal of flow (hepatofugal - *directed away from the liver*). As an aside, apparently “fugal” is latin for “flee.” So the blood is fleeing the liver, or running away from it. Reversed flow in the portal system is seen in cirrhosis between 5-25%. Why you might ask, does the portal vein reverse flow instead of just clotting off in the setting of high resistance to inflow? The answer has to do with the unique dual hepatic blood supply (70 P / 30 A). As mentioned above, in cirrhosis, the principal area of obstruction to blood flow is believed to be in the outflow vessels (the hepatic venules and distal sinusoids). The outflow obstruction also affects the hepatic artery, causing increased resistance as well. So why doesn't the artery clot or reverse ? The difference is that the portal system can decompress through the creation of collaterals, and the artery cannot. So the artery does something else, it opens up tiny little connections to the portal system. The enlargement of these tiny communications has been referred to as “parasitizing the portosystemic decompressive apparatus.” If the resistance is high enough, hepatic artery inflow will be shunted into - and can precipitate hepatofugal flow in the portal vein. So, in patients with hepatofugal flow in the main portal vein or intrahepatic portal vein branches, the shunted blood comes from the hepatic artery.

With increased resistance in the liver to the portal circulation, you also start to have colonic venous stasis (worse on the right). This can lead to “*Portal Hypertensive Colopathy*”, which is basically an edematous bowel that mimics colitis. Why is it worse on the right? The short answer is that collateral pathways develop more on the left (splenorenal shunt, short gastrics, esophageal varices), and decompress that side. The trivia question is that it does resolve after transplant. The same process can affect the stomach “*Portal Hypertensive Gastropathy*” causing a thickened gastric wall on CT, and well as cause upper GI bleeding in the absence of varices.

Earlier I mentioned that hepatocytes react to injury by turning into regenerative nodules. This is how multi-focal HCC starts. Regenerative nodules -> Dysplastic nodules (increased size and cellularity) -> HCC. As this process takes place, the nodule changes from preferring to drink portal blood to only wanting to drink arterial blood. This helps explain why HCC has arterial enhancement and rapid washout. The transformation also follows a progression from T2 dark (regenerative) -> T2 bright (HCC). A buzzword is “*nodule within nodule*” where a central bright T2 nodule, has a T2 dark border. This is concerning for transformation to HCC.

Regenerative	Dysplastic	HCC
Contains Iron	Contains Fat, Glycoprotein	
T1 Dark, T2 Dark	T1 Bright, T2 Dark	T2 Bright
Does NOT Enhance	Usually Does NOT Enhance	Does Enhance

There is one last concept, that I wanted to squeeze in. The squeezing that causes portal hypertension, also squeezes out most benign liver lesions (cysts, hemangiomas). So, lesions in a cirrhotic liver should be treated with more suspicion.

Congenital

Cystic Kidney Disease (both AD and AR): Patient's with AD polycystic kidney disease will also have cysts in the liver. This is in contrast to the AR form in which the liver tends to have fibrosis.



Hereditary Hemorrhagic Telangiectasia (Osler-Weber-Rendu) Autosomal dominant disorder characterized by multiple AVMs in the liver and lungs. It leads to cirrhosis, and a massively dilated hepatic artery. The other high yield trivia is that the lung AVMs set you up for brain abscess.

Infection

Infection of the liver can be thought of as either viral, abscess (pyogenic or amoebic), fungal, parasitic, or granulomatous.

Viral: Hepatitis which is chronic in B and C, and acute with the rest. A point of trivia is the **HCC in the setting of hepatitis can occur in the acute form of Hep B** (as well as chronic). Obviously, chronic hep C increases risk for HCC. On ultrasound the “starry sky” appearance can be seen. Although, this is non-specific and basically just the result of liver edema making the fat surrounding the portal triads look brighter than normal.

Infection Buzzwords	
Viral Hepatitis	Starry Sky
Pyogenic Abscess	Double Target
Candida	Bull's Eye
Amoebic Abscess	“Extra Hepatic Extension”
Hydatid Disease	Water Lilly, Sand Storm
Schistosomiasis	Tortoise Shell

Pyogenic: These can mimic cysts. For the purpose of multiple choice, *a single abscess is Klebsiella, and multiple are E. Coli*. The presence of gas is highly suggestive of pyogenic abscess. “Double Target” sign with central low density, with rim enhancement, surrounded by more low density can be seen with CT. If the amebic abscess is in the left lobe, it needs to be emergently drained (can rupture into the pericardium).

Liver Masses:

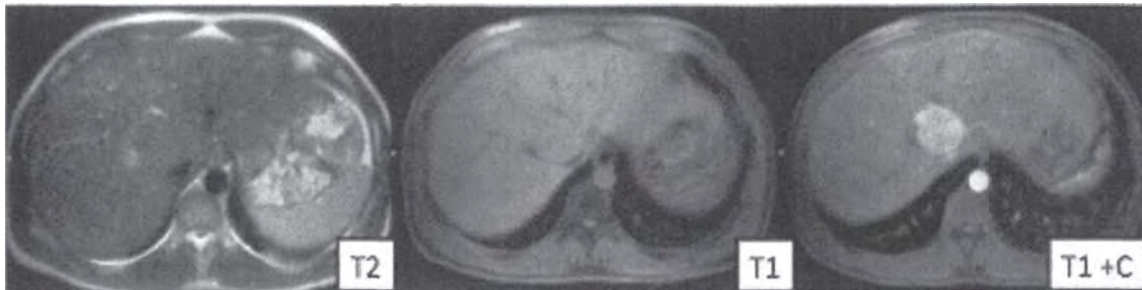
Hemangioma: This is the most common benign liver neoplasm. Favors women 5:1. They may enlarge with pregnancy. On US will be bright (unless it’s in a fatty liver, than can be relatively dark). On US, flow can be seen in vessels adjacent to the lesion but NOT in the lesion. On CT and MRI tends to match the aorta in signal and have “peripheral nodular discontinuous enhancement”. Should totally fill in by 15 mins. Atypical hemangioma can have the “reverse target sign.”

Trivia: A hemangioma can change its sonographic appearance during the course of a single examination. No other hepatic lesion is known to do this.

Hemangioma US Pearls:

- * Need to core for biopsy, FNA does not get enough tissue (only blood)
- * Hyperechoic (65%)
- * Enhanced thru transmission is common
- * NO Doppler flow inside the lesion itself
- * Atypical appearance - hyperechoic periphery, with hypoechoic center (inverse target)
- * Calcifications are extremely rare

Focal Nodular Hyperplasia (FNH) Believed to start in utero as an AVM. This is the second most common benign liver neoplasm . It is NOT related to OCP use. It is composed of normal hepatocytes, abnormally arranged ducts, and Kupffer cells (reticuloendothelial cells). May show spoke wheel on US Doppler. On CT, should be “homogenous” on arterial phase. Can be a “**Stealth**” lesion on MRI - T1 and T2 isointense. Can have a central scar. Scar will demonstrate delayed enhancement (like scars do). *Biopsy Trivia: You have to hit the scar, otherwise path results will say normal hepatocytes.* Sulfur Colloid is always the multiple choice test question (reality is that its only hot 30-40%). Unlike hepatic adenomas, they are not related to the use of birth control pills, although as a point of confusing trivia and possibly poor multiple choice test question writing, birth control pills may promote their growth.



FNH - The "Stealth Lesion" - Iso on T1 and T2

This vs That: Central Scars of FNH and Fibrolamellar HCC	
FNH	FL HCC
T2 Bright	T2 Dark (usually)
Enhances on Delays	Does NOT enhance
Mass is Sulfur Colloid Avid (sometimes)	Mass is Gallium Avid

Hepatic Adenoma: Usually a solitary lesion seen in a female on OCPs. Alternatively could be seen in a man on anabolic steroids. When it's multiple you should think about glycogen storage disease (von Gierke) or liver adenomatosis. No imaging methods can reliably differentiate hepatic adenoma from hepatocellular carcinoma. Rarely, they *may degenerate into HCC* after a long period of stability. They *often regress after OCPs are stopped*. Their propensity to bleed sometimes makes them a surgical lesion if they won't regress.

Trivia:

Q: Most common location for hepatic adenoma (75%)

A: Right Lobe liver

Management: You stop the OCPs and re-image, they should get smaller. Smaller than 5cm, watch them. Larger than 5cm they often resect because (1) they can bleed and (2) they can rarely turn into cancer.

This vs That: Hepatic Adenoma vs FNH	
Hepatic Adenoma	FNH
Usually > 8cm	Usually < 8cm
No Bile Ducts	Normal Bile Ducts
No Kuplfer Cells	Normal Kupffer Cells
Sulfur Colloid Cold	Sulfur Colloid Hot (sometimes)

HCC Occurs typically in the setting of cirrhosis and chronic liver disease; Hep B, Hep C, hemochromatosis, glycogen storage disease, Alpha 1 antitrypsin. AFP elevated in 80-95% Will often invade the portal vein, although invasion of the hepatic vein is considered a more “specific finding.”

“*Doubling Time* ” - *the classic Multiple Choice Question*. This is actually incredibly stupid to ask because there are 3 described patterns of growth (slow, fast, and medium). To make it an even worse question, different papers say different stuff. Some say: Short is 150 days, Medium to 150-300, and Long is >300. I guess the answer is 300 - because it’s in the middle. Others define medium at around 100 days. A paper in Radiology {*May 2008 Radiology, 247, 311-330*) says 18-605 days. The real answer would be to say follow up in 3-4.5 months.

Other Random Trivia: HCCs like to explode and cause spontaneous hepatic bleeds.

Fibrolamellar Subtype of HCC: This is typically seen in a younger patients (<35) without cirrhosis and a normal AFP. The buzzword is central scar. The scar is similar to the one seen in FNH with a few differences. This scar does NOT enhance, and is T2 Bright. As a point of trivia, this tumor is Gallium avid. This tumor calcifies more often than conventional HCC.

This vs That: HCC vs Fibrolamellar Subtype HCC	
HCC	FL HCC
Cirrhosis	No Cirrhosis
Older (50s-60s)	Young (30s)
Rarely Calcifies	Calcifies Sometimes
Elevated AFP	Normal AFP

Cholangiocarcinoma: Where HCC is a cancer of the hepatocyte, cholangiocarcinoma is a cancer of the bile duct. It is usually seen in an elderly (70s) man. There are multiple risk factors (**Primary sclerosing cholangitis**, recurrent pyogenic cholangitis, clonorchis senesis (the liver fluke), HIV, Hep B&C, EtOH, and of course thorotrast). Primary sclerosing cholangitis is the major risk factor in western countries. Just like a pancreatic head cancer the buzzword is “painless jaundice.”

These tumors have an infiltrative growth pattern, and will not have a capsule. On imaging it will show dilation of the biliary system, and possible persistent enhancing soft tissue on delayed phase (the scar enhances). **Capsular retraction is a buzzword**, mainly for the mass forming subtype. Encasement of a portal or hepatic vein without formation of a visible tumor thrombus is one of the distinguishing features of cholangiocarcinoma versus HCC.

Key Findings:

- * *Delayed Enhancement*
- * *Peripheral biliary dilation*
- * *Capsular Retraction*

Klatskin Tumor: A Klatskin tumor is a type of cholangiocarcinoma that occurs at the bifurcation of the right and left hepatic ducts. The tumor has dense fibrosis (which enhances on delayed imaging).

Hepatic Angiosarcoma: This used to be the go to for thorotrast questions. Even though everyone who got thorotrast died 30 years ago, a few dinosaurs writing multiple choice test questions still might ask it. Hepatic Angiosarcoma is very rare, although technically the most common primary sarcoma of the liver. It is associated with toxic exposure - arsenic use (latent period is about 25years), Polyvinyl chloride exposure, Radiation, and yes... thorotrast. Additional trivia, is that you can see it in Hemochromatosis and NF patients.

It's usually multifocal, and has a propensity to bleed.

Biliary Cystadenoma Uncommon benign cystic neoplasm of the liver. Usually seen in middle aged women. Can sometimes present with pain, or even jaundice. They can be unilocular or multilocular and there are no reliable methods for distinguishing from biliary cystadenocarcinoma (which is unfortunate).

Mets to the Liver: If you see mets in the liver first think colon. Calcified mets are usually the result of a mucinous neoplasm (colon, ovary, pancreas).

With regard to ultrasound: Hyperechoic mets are often hypervascular (renal, melanoma, carcinoid, choriocarcinoma, thyroid, islet cell). Hypoechoic mets are often hypovascular (colon, lung, pancreas).

"Too Small Too Characterize" - even in the setting of breast cancer (with no definite hepatic mets) tiny hypodensities have famously been shown to be benign 90-95% of the time.

Lymphoma: Hodgkins lymphoma involves the liver 60% of the times (Non Hodgkins is around 50%), and may be hypoechoic.

Kaposi Sarcoma: Seen in patient's with AIDS. Causes diffuse periportal hypoechoic infiltration. Looks similar to biliary duct dilation.

Liver Contrast Timing

Few Quick points on timing of contrast (this lends well to multiple choice).

- Arterial Phase (Hepatic) - 25-30 seconds
- Portal Venous Phase - 70 seconds

Sulfur Colloid HOT or COLD		
Hepatic Adenoma	COLD	
FNH	40% HOT, 30% COLD, 30% Warm	
Cavernous Hemangioma	COLD	RBC Scan HOT
HCC	COLD	Gallium HOT
Cholangiocarcinoma	COLD	
Mets	COLD	
Abscess	COLD	Gallium HOT
Focal Fat	COLD	Xenon HOT

Benign Liver Masses					
	Ultrasound	CT	MR	Trivia	
Hemangioma	Hyperechoic with increased though transmission	Peripheral Nodular Discontinuous Enhancement	T2 Bright	Rare in Cirrhotics	
FNH	Spoke Wheel	Homogenous Arterial Enhancement	“Stealth Lesion - Iso on T1 and T2”	Central Scar	Bright on Delayed Eovist (Gd-EOB-DTPA)
Hepatic Adenoma	Variable	Variable	Fat Containing on In/Out Phase	OCP use, Glycogen Storage Disease	Can explode and bleed
Hepatic Angiomyolipoma	Hyperechoic	Gross Fat	T1/T2 Bright	Unlike renal AML, 50% don't have fat	Tuberous Sclerosis

Diffuse Liver Processes:

Fatty Liver: Very common in America. Can be focal (next to gallbladder or ligamentum teres), can be diffuse, can be diffuse with sparing. You can call it a few different ways. Two standard deviation difference between in and out of phase imaging (lower on out of phase). For CT, if it's a non-contrast study 40 H.U. is a slam dunk. If it's contrasted some people say you can NEVER call it. Others say it's ok if (a) it's a good portal venous phase (b) the H.U. is less than 100, and (c) it's 25 H.U. less than the spleen. On US, if the liver is brighter than the right kidney you can call it. Hepatosteatorosis is a fat liver. NASH (hepatitis from a fat liver) has abnormal LFTs.

What causes it? McDonalds, Burger King, and Taco Bell. Additional causes include chemotherapy (breast cancer), steroids, cystic fibrosis.

Budd Chiari Syndrome Classic multiple choice scenario is a pregnant woman, but can occur in any situation where you are hypercoagulable (*most common cause is idiopathic*). The result of hepatic vein thrombosis.

The characteristic findings of Budd-Chiari syndrome include hepatic venous outflow obstruction, intrahepatic and systemic collateral veins, and large regenerative (hyperplastic) nodules in a dysmorphic liver. The caudate lobe is often massively enlarged (spared from separate drainage into the IVC). In the acute phase, the liver will show the classic ‘flip-flop pattern’ on portal phase with low attenuation central, and high peripherally. The liver has been described as “nutmeg” with an inhomogeneous mottled appearance, and delayed enhancement of the periphery of the liver.

Who gets a “nutmeg liver” ???

- *Budd Chiari*
- *Hepatic Veno-occlusive disease*
- *Right Heart Failure (Hepatic Congestion)*
- *Constrictive Pericarditis*

Regenerative (hyperplastic) nodules are very difficult to distinguish from multifocal hepatocellular carcinoma. They are bright on both T1 and T2. Multiple big (>10cm) and small (<4cm) nodules in the setting of Budd-Chiari suggest a benign process.

Presentation can be acute or chronic. Acute from thrombus into the hepatic vein or IVC. These guys will present with **rapid onset ascites**. Chronic from fibrosis of the intrahepatic veins, presumably from inflammation.

Who gets massive caudate lobe hypertrophy???

- *Budd Chiari*
- *Primary Sclerosing Cholangitis*
- *Primary Biliary Cirrhosis*

Hepatic Veno-occlusive Disease: This is a form of Budd Chiari that occurs from occlusion of the small hepatic venules. It is endemic in Jamaica (from Alkaloid bush tea). In the US it's typically the result of XRT and chemotherapy. The main hepatic veins and IVC will be patent, but portal waveforms will be abnormal (slow, reversed, or to-and fro).

Hemochromatosis: They can show this two main ways. The first is just liver and spleen being T1 and T2 dark. The second (and more likely) way this will be shown is in and out of phase changes the opposite of those seen in hepatic steatosis. **Low signal on in phase, and high signal on out of phase.**

The second main piece of trivia is to tell *primary vs secondary*. Primary is the inherited type, caused by more GI uptake, with resulting iron overload. The key point is the pancreas is involved and the spleen is spared. Secondary is the result of either chronic inflammation or multiple transfusions. The body reacts by trying the “Eat the Iron”, with the reticuloendothelial system. The key point is the pancreas is spared and the spleen is not. **“Primary = Pancreas”, “Secondary = Spleen”**

Hemochromatosis	
Primary	Secondary
Genetic - increased absorption	Acquired - chronic illness, and multiple transfusions
Liver, Pancreas	Liver, Spleen
Heart, Thyroid, Pituitary	

Additional trivia to know is that hemochromatosis is a major HCC *{and colorectal CA}* risk factor with 1/3 of the deaths resulting from HCC. The step one question was “bronze diabetes” from pancreas damage in primary hemochromatosis.

Lastly hemochromatosis is in the dense liver DDx.

Dense Liver DDx:

- *Hemochromatosis*
- *Wilson s Disease*
- *Colloidal Gold Therapy*
- *Amiodarone*

Passive Congestion: Passive hepatic congestion is caused by stasis of blood within the liver due to compromise of hepatic drainage. It is a common complication of congestive heart failure and constrictive pericarditis. It is essentially the result of elevated CVP transmitted from the right atrium to the hepatic veins.

Findings include:

- *Refluxed contrast into the hepatic veins*
 - *Increased portal venous pulsatility*
- ' Nutmeg liver*

Misc Liver Conditions:

Portal Vein Thrombosis: Occurs in hypercoagulable states (cancer, dehydration, etc...)- Can lead to cavernous transformation. with the development of a bunch of serpiginous vessels in the porta hepatis which may reconstitute the right and left portal veins. This takes like 12 months to happen (*it proves portal vein is chronically occluded*).

Pseudo Cirrhosis: Treated breast cancer mets to the liver can cause contour changes that mimic cirrhosis. Specifically multifocal liver retraction and enlargement of the caudate has been described. Why this is specific for breast cancer is not currently known, as other mets to the liver don't produce this reaction.

Cryptogenic Cirrhosis: Essentially cirrhosis of unknown cause. Most of these cases are probably the result of nonalcoholic fatty liver disease.

Liver Transplant: The liver has great ability to regenerate and may double in size in as little as 3 weeks making it ideal for partial donation. Hepatitis C is the most common disease requiring transplantation (followed by EtOH liver disease, and cryptogenic cirrhosis). In adults, right lobes (segments 5-8) are most commonly implants. This is the opposite of pediatric transplants, which usually donates segments 2-3. The modern surgery has four connections (IVC, artery, portal vein, CBD).

Contraindications include, extrahepatic malignancy, advanced cardiac disease, advanced pulmonary disease, or active substance abuse, Portal HTN is NOT a true contraindication although it does increase the difficulty of the surgery and increase mortality.

Normal Transplant US

- Normal Doppler should have a **RAPID systolic upstroke**
 - Diastolic -> Systolic in less than 80msec (0.08 seconds)
- Resistive Index is Normally between 0.5-0.7
- Hepatic Artery Peak Velocity should be < 200 cm/sec

Syndrome of Impending Thrombosis

- 3-10 days post transplant
- (1st) Initial Normal Waveform
- (2nd) No diastolic flow
- (3rd) Dampening Systolic flow
 - Tardus Parvus
 - RI < 0.5
- (4th) Loss of Hepatic Waveform

As mentioned before, the normal liver gets 70% blood flow from the portal vein, making it the key player. In the transplanted liver, the hepatic artery is the king and is the primary source of blood flow for the bile ducts (which undergo necrosis with hepatic artery failure). Hepatic artery thrombosis comes in two flavors: early (<15 days), and later (years). The late form is associated with chronic rejection and sepsis.

Trivia: Tardus Parvus is more likely secondary to stenosis than thrombosis

Biliary

Jaundice: You always think about common duct stone, but the most common etiology is actually from a benign stricture (post traumatic from surgery or biliary intervention).

Bacterial Cholangitis: Hepatic abscess can develop secondary to cholangitis, usually as the result of stasis (so think stones). The triad of jaundice, fever, and right upper quadrant pain is the step 1 question.

Primary Sclerosing Cholangitis (PSC) Chronic cholestatic liver disease of unknown etiology characterized by progressive inflammation which leads to multifocal strictures of the intra and or extrahepatic bile ducts. The disease often results in cirrhosis, and is **strongly associated with cholangiocarcinoma**. The buzzword for the **cirrhotic pattern** is “**central regenerative hypertrophy**”. It is associated with inflammatory bowel disease (Ulcerative Colitis 80%, Crohns 20%). It is an indication for transplant, with a post transplant recurrence of about 20%.

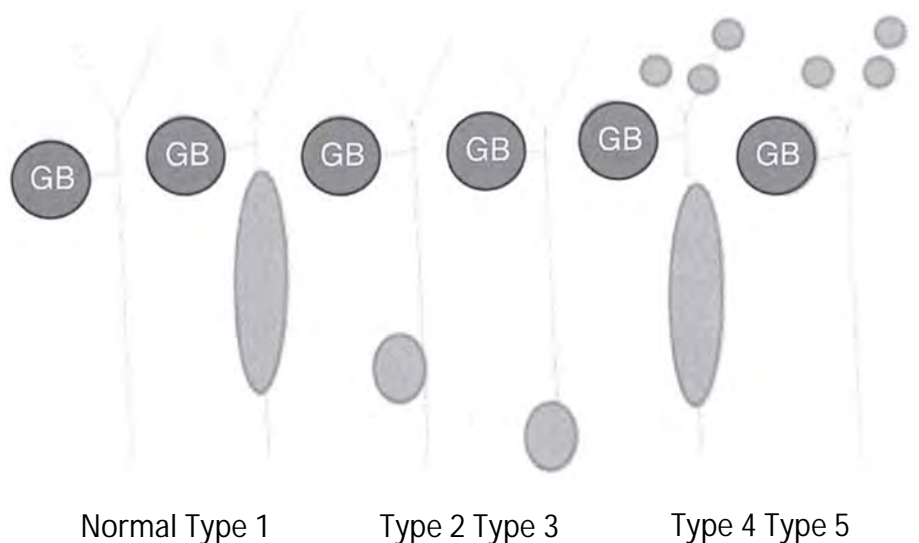
AIDS Cholangiopathy: Infection of the biliary epithelium (*classically Cryptosporidium*) can cause ductal disease in patients with AIDS. The appearance mimics PSC with intrahepatic and/or extrahepatic multifocal strictures. The classic multiple choice test question is the association with papillary stenosis (which occurs 60% of the time).

This vs That: AIDS Cholangiopathy vs Primary Sclerosing Cholangitis	
AIDS	PSC
Focal Strictures of the extrahepatic duct > 2cm	Extrahepatic strictures rarely > 5mm
Absent saccular deformities of the ducts	Has saccular deformities of the ducts
Associated Papillary Stenosis	

Oriental Cholangitis (*Recurrent pyogenic cholangitis*): Common in Southeast Asia (hence the name). They always show it as **dilated ducts, that are full of pigmented stones**. A buzzword is “straight rigid intrahepatic ducts.” The cause of the disease is not known, but it may be associated with clonorchiasis, ascariasis, and nutritional deficiency . These guys don’t do as well with endoscopic decompression, and often need surgical decompression.

Primary Biliary Cirrhosis: An autoimmune disease that results in the destruction of small bile ducts. It primarily affects **middle aged women**, who are often asymptomatic. In the early disease, normal bile ducts help distinguish it from PSC. In later stages, there is irregular dilation of the intrahepatic ducts, with **normal extrahepatic ducts**. There is increased risk of HCC. If caught early it has an excellent prognosis and responds to medical therapy with ursodexychoic acid. The step 1 trivia is “**antimitochondrial antibodies** (AMA)” which are present 95% of the time.

Choledochal Cysts / Caroli’s: Choledochal cysts are congenital dilation of the bile ducts - classified into 5 types by some dude named Todani. *The high yield trivia is type 1 is focal dilation of the CBD and is by far the most common.* Type 2 and 3 are super rare. Type 2 is basically a diverticulum of the bile duct. Type 3 is a “choledochocele.” Type 4 is both intra and extra. Type 5 is Caroli’s, and is intrahepatic only.



Caroli’s is an AR disease associated with polycystic kidney disease and medullary sponge kidney. The hallmark is intrahepatic duct dilation, that is large and sacular. **Buzzword** is “**central dot sign**” which corresponds to the portal vein surrounded by dilated bile ducts.

Complications

- * **Cholangiocarcinoma**
- * **Cirrhosis**
- * **Cholangitis**
- * **Intraductal Stones**

Gall Bladder

Normal Gallbladder

The normal gallbladder is found inferior to the interlobar fissure between the right and left lobe. The size varies depending on the last meal, but is supposed to be $<4 \times <10\text{cm}$. The wall thickness should be $<3\text{mm}$. The lumen should be anechoic.

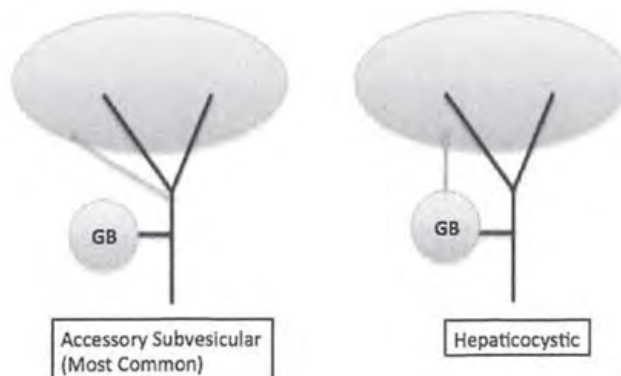
Variants / Congenital

Phyringian Cap: A phyringian cap is seen when the GB folds on itself. It means nothing.

Intrahepatic Gallbladder: Variations in gallbladder location are rare, but the intrahepatic gallbladder is probably the most frequently recognized variant. Most are found right above the interlobar fissure.

Duplicated Gallbladder: It can happen.

Duct of Luschka: An accessory cystic duct. This can cause a big problem (persistent bile leak) after cholecystectomy. There are several subtypes which is not likely to be tested.



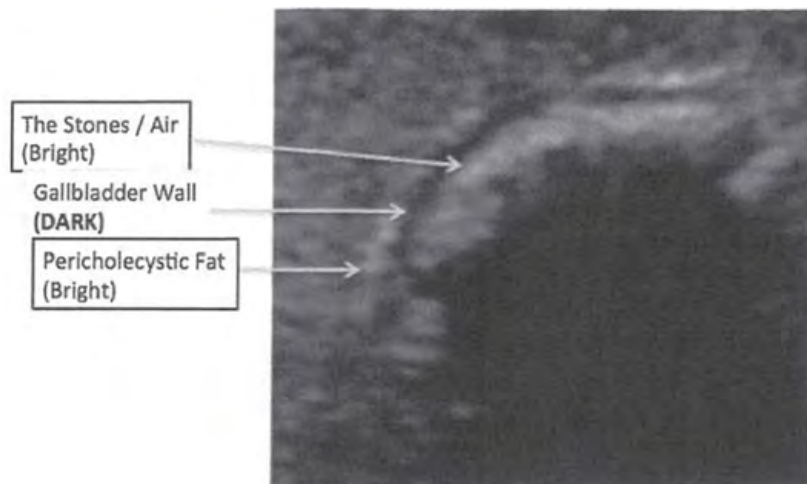
Wall Thickening: Very non-specific. Can occur from biliary (Cholecystitis, AIDS, PSC...) or non-biliary causes (hepatitis, heart failure, cirrhosis, etc....).

Gallstones: Gallstones are found in 10% of asymptomatic patients/ Most (75%) are cholesterol, the other 25% are pigmented. They cast shadows.

Reasons a stone might not cast a shadow

- *It's not a stone*
- *It's a stone, but $< 3\text{mm}$ in size*
- *The sonographer sucks*

(WES) Wall Echo Shadow GB



Can occur for three reasons.

- (1) Gallbladder full of stones * Clean shadowing.*
- (2) Porcelain Gallbladder ^Variable shadowing*
- (3) Emphysematous Cholecystitis *Dirty shadowing.*

Porcelain Gallbladder: Extensive wall calcification. The key point is increased risk of GB Cancer. These are surgically removed.

Gallbladder Polyps: These can be cholesterol (by far the most common), or not cholesterol (adenomas, papillomas). Cholesterol polyps aren't real polyps, but instead are essentially enlarged papillary fronds full of lipid filled macrophages, that are attached to the wall by a stalk.

The non-cholesterol subtypes are almost always solitary and are typically larger. The larger polyps may have Doppler flow. They are NOT mobile, and do NOT shadow. *Once they get to be lcm , people start taking them out.*

Adenomyomatosis: Results from hyperplasia of the wall with formation of intramural mucosal diverticula (Rokitansky-Aschoff sinuses) which penetrate into the wall of the gallbladder. These diverticula become filled with cholesterol crystals - which manifest from the unique acoustic signature as comet-tail artifact (highly specific for adenomyomatosis).

Comes in 3 flavors: Generalized (diffuse), Segmental (annular), and Fundal (localized or adenomyoma). The Localized form can't be differentiated from GB cancer.

Gallbladder Cancer: Key points are that most GB cancers are associated with gallstones. The outcomes are terrible with 80% having direct tumor invasion of the liver or portal nodes at the time of diagnosis.

Mirizzi Syndrome: This occurs when the common hepatic duct is obstructed secondary to an impacted cystic duct stone. The stone can eventually erode into the CHD or GI tract. Key point is the increased co-incidence of gallbladder CA (5x more risk) with Mirizzi. Another key piece of trivia is that Mirizzi occurs more in people with a low cystic duct insertion (normal variant), allowing for a more parallel course and closer proximity to the CHD.

Doppler of the Liver

Brief introduction to terminology.

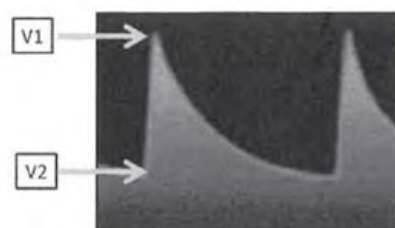
- * “Duplex ” means color.
- * “Spectral ” means color with a waveform.

Concept of Arterial Resistance:

Some organs require continuous flow (brain), whereas others do not (muscles). The body takes advantage of this to preserve energy. When an organ needs to be “on,” its arteriolar bed dilates, and the waveform becomes low resistance, and the organ is appropriately perfused. When an organ goes to “power save” mode, its arterioles constrict, the waveform switches to high resistance, and flow is diverted to other more vital organs.

To help quantify this low resistance high resistance thing. We use this “Resistive Index (RI)” - which is defined as $V1-V2/V1$.

Just remember that things that need blood all the time, will have continuous diastolic flow - and thus a low resistance wave form.



What does RI mean in the Liver:

- * The “normal” RI in the liver is between 0.5 and 0.7.

What if it's >0.7 (too high) ?

- * Basically, a high RI (> 0.7) doesn't mean shit as an isolated finding. A high RI is not specific for liver disease. An RI that is too high may be the result of the postprandial state, advanced patient age, or diffuse distal microvascular disease, which has a wide variety of causes including chronic liver disease from cirrhosis or chronic hepatitis.

What if it's <0.5 (too low) ?

- * A low RI is either the result of proximal stenosis or distal vascular shunting. The most common cause of this shunting is the arteriovenous or arteriportal fistulas seen in severe cirrhosis. Other more rare causes would be trauma (including iatrogenic injury - liver biopsy) or total zebras like Osier-Weber-Rendu syndrome.

Causes of Low RI (< 0.5)		Causes of High RI (> 0.7)
Proximal Arterial Narrowing	Peripheral Vascular Shunts	Postprandial
Atherosclerotic Disease (Celiac, or Hepatic)	Shunting seen with Cirrhosis	Advanced Age
Stenosis at an anastomosis (transplant)	Post Traumatic (liver biopsy)	Cirrhosis
Median Arcuate Ligament Compression (severe)	Osier-Weber-Rendu	Hepatic Congestion (Acute or Chronic)
		Transplant Rejection

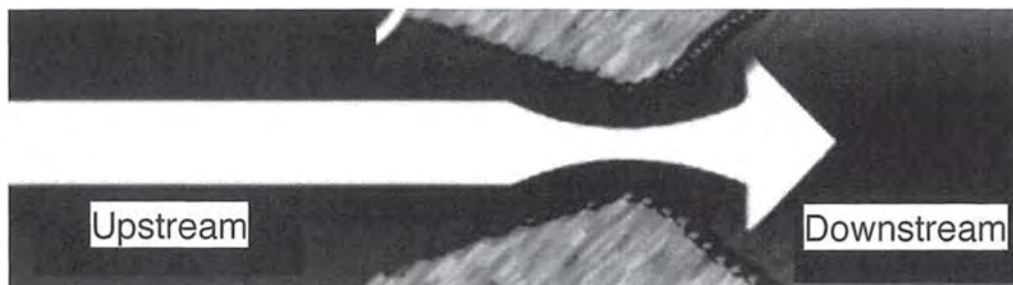
How can Cirrhosis cause low RI and high RI?

- * Essentially, the shunts that develop decrease RI, BUT the fibrosis that develops increases RI. So, it's a balance between these two things. As a result, they may be high, normal, or low - and RI is NOT useful for diagnosing cirrhosis or predicting how severe it is.

Understanding Stenosis:

The vocabulary of “Upstream vs Downstream” is somewhat confusing. Try and remember, that the flow of blood defines the direction.

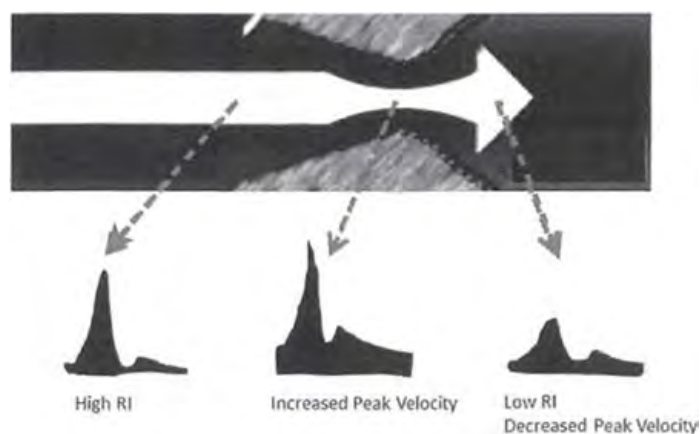
- * Upstream = Blood that has NOT yet passed through the stenosis
- * Downstream = Blood that has passed area of stenosis



So there are direct signs, and indirect signs of stenosis.

Direct Signs: The direct signs, are those found at the stenosis itself and they include elevated peak systolic velocity, and spectral broadening (immediate post stenotic).

Indirect Signs: The indirect signs are going to be tardus parvus (downstream) - with time to peak < 70msec. The RI downstream will be low (< 0.5), because the liver is starved for blood. The RI upstream will be elevated (> 0.7) because that blood needs to overcome the area of stenosis.

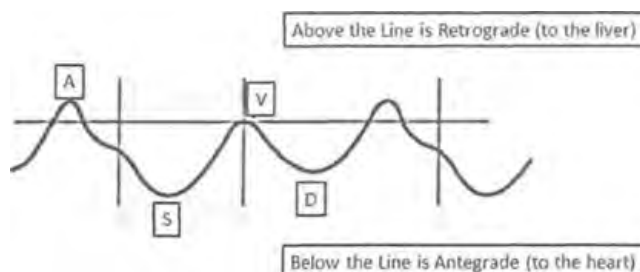


Hepatic Veins

Flow in the hepatic veins is complex, with alternating forward and backward flow. The bulk of the flow should be forward “antegrade” (liver → heart). Things that mess with the waveform are going to be pressure changes in the right heart are transmitted to the hepatic veins (CHF, Tricuspid Regurg) or compression of the veins directly (cirrhosis).

Anything that increases right atrial pressure (atrial contraction) will cause the wave to slope upward. “A” represents atrial contraction.

Anything that decreases right atrial pressure will cause the wave to slope downward.



Abnormal Hepatic Vein Waveforms can manifest in one of three main categories: (1) More pulsatile, (2) Less Pulsatile, (3) Absent - Budd Chiari.

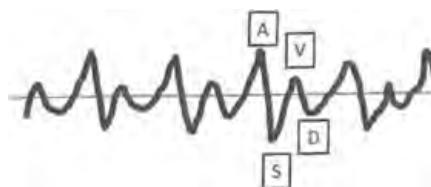
Increased HV Pulsatility	Decreased HV Pulsatility
Tricuspid Regurg	Cirrhosis
Right Sided CHF	Hepatic Venous Outflow Obstruction (any cause)

An extra credit trick is to contrast the waveforms of Tricuspid Regurg vs Right Sided CHF (I’d consider this low yield for the CORE, but is a classic piece of trivia).

D wave Deeper than S wave = Tricuspid Regurg



Tricuspid Regurg
-Increased Pulsatility
-Very tall “V”
-D wave deeper than S



Right Sided CHF
-Increased Pulsatility
-Normal D and S relationship

Portal Vein

Flow in the portal vein should always be towards the liver (antegrade). You can see some normal cardiac variability from hepatic venous pulsatility transmitted through the hepatic sinusoids. Velocity in the normal hepatic vein is between 20-40 cm/s. The waveform should be a gentle undulation, always remaining above the baseline.

You have three main patterns:

- (1) Normal
- (2) Pulsatile
- (3) Reversed



Cause of Portal Vein Pulsatility: **Right sided CHF, Tricuspid Regurg, Cirrhosis** with Vascular AP shunting.

Causes of Portal Vein Reversed Flow: The big one is **Portal HTN** (any cause).

Absent Flow: This could be considered a fourth pattern. It's seen in thrombosis, tumor invasion, and stagnant flow from terrible portal HTN.

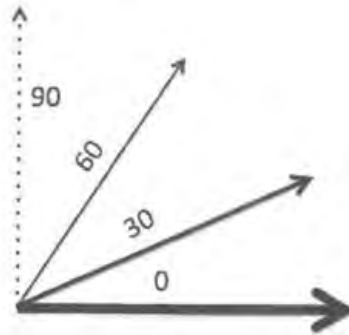
Slow Flow: Velocities less than 15 cm/s. Portal HTN is the most common cause. Additional causes are grouped by location: *

- * Pre - Portal Vein Thrombosis
- * Intra - Cirrhosis (any cause)
- * Post - Right sided Heart Failure, Tricuspid Regurg, Budd-Chiari

Final Doppler Trivia:

An ultra common quiz question is to ask “**what should the Doppler angle be?**” Now even though ultrasound physics is covered in more detail in the dedicated section this is a high yield enough point to warrant repetition. **The answer is “less than 60.”**

Why? If you look at the; $\cos 90 = 0$, $\cos 60 = 0.5$, $\cos 0 = 1.0$ - the doppler strength follows the Cos.



The Pancreas

Trivia regarding the pancreas can be broadly clustered into: Solid Lesions, Cystic Lesions, Pancreatitis, and Misc Trivia (mostly developmental stuff).

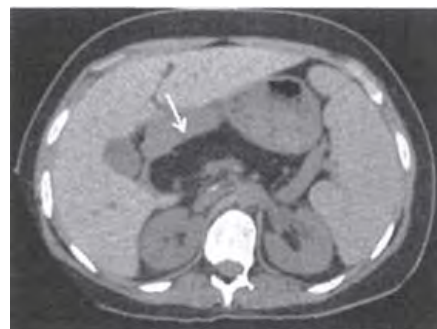
Misc Pancreas Trivia:

Anatomy: The pancreas is a retroperitoneal structure (the tail may be intraperitoneal).

Cystic Fibrosis: The pancreas is affected in 85-90% of CF patients. Inspissated secretions cause proximal duct obstruction leading to the two main changes in CF: (1) Fibrosis (decreased T1 and T2 signal) and the more common one (2) fatty replacement (increased T1). Patient's with CF, who are diagnosed as adults, tend to have more pancreas problems than those diagnosed as children. Those with residual pancreatic exocrine function can have bouts of recurrent acute pancreatitis. Small (1-3mm) pancreatic cysts are common.

High Yield Trivia:

- *Complete fatty replacement is the most common imaging finding in adult CF*
- *Markedly enlarged with fatty replacement has been termed lipomatous pseudohypertrophy of the pancreas. *This is a buzzword.*
- *Fibrosing Colonopathy: Wall thickening of the proximal colon as a complication of enzyme replacement therapy.*



CF- Marked Fatty Replacement of Pancreas



Shwachman-Diamond Syndrome: The 2nd most common cause of pancreatic insufficiency in kids (CF #1). Basically, it's a kid with diarrhea, short stature (metaphyseal chondroplasia), and eczema. *Will also cause lipomatous pseudohypertrophy of the pancreas.*

Pancreatic Lipomatosis: Most common pathologic condition involving the pancreas. The most common cause in childhood is CF. Additional causes worth knowing are Cushing Syndrome, Chronic Steroid Use, Hyperlipidemia, and Shwachman-Diamond Syndrome.

This vs That: Pancreatic Agenesis vs Pancreatic-Lipomatosis	
Agenesis	Lipomatosis
Does NOT have a duct	Does have a duct

Annular Pancreas: Essentially an embryologic screw up (*failure of ventral bud to rotate with the duodenum*), that results in encasement of the duodenum. Results in a rare cause of duodenal obstruction (10%), that typically presents as duodenal obstruction in children and pancreatitis in adults. Can also be associated with other vague symptoms (post-prandial fullness, “symptoms of peptic ulcer disease”, etc...).

- * Can be Complete or Incomplete: When it's incomplete the term “crocodile jaw appearance” is a buzzword.
- * Can be extramural or intramural: Extramural has pancreatic tissue surrounding the duodenum (*<drains into the main duct*). Intramural has pancreatic tissue in the wall of the duodenum (*drains via small ducts into the lumen*).
- * On imaging, look for an annular duct encircling the descending duodenum.



Annular Pancreas
- Encircling the duodenum

Dorsal Pancreatic Agenesis - All you need to know is that (1) this sets you up for diabetes (*most of your beta cells are in the tail*), and (2) it's associated with polysplenia.

Pancreatic Trauma: The pancreas sits in front of the vertebral body, so it's susceptible to getting smashed in blunt trauma. Basically, **the only thing that matters is integrity of the duct**. If the duct is damaged they need to go to the OR. The most common delayed complication is pancreatic fistula (10-20%), followed by abscess formation. Signs of injury can be subtle, and may include focal pancreatic enlargement, or adjacent stranding/fluid.

Suspected Pancreatic Duct Injury? - Next Step - MRCP or ERCP

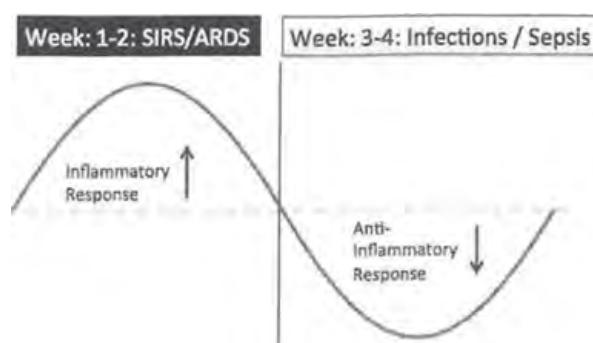
Pancreatitis:

Acute Pancreatitis:

Etiology: By far the most common causes are gallstones and EtOH which combined make up 80% of the cases in the real world. However, for the purpose of multiple choice tests a bite from the native scorpion of the island of Trinidad and Tobago is more likely to be the etiology. Additional causes include, ERCP (*which usually results in a mild course*), medications (*classically valproic acid*), trauma (*the most common cause in a child*), pancreatic cancer, infectious (*post viral in children*), hypercalcemia, hyperlipidemia, autoimmune pancreatitis, pancreatic divisum, groove pancreatitis, tropic pancreatitis, and parasite induced.

Clinical Outcomes: Prognosis can be estimated with the "Balthazar Score." Essentially, you can think about pancreatitis as "mild" (*no necrosis*) or "severe" (*having necrosis*). Patient's with necrosis don't start doing terrible until they get infected, then the mortality is like 50-70%.

Severe Pancreatitis: Severe acute pancreatitis has a biphasic course. With the first two weeks being a pro-inflammatory phase. This is a sterile response in which infection rarely occurs. The third and fourth weeks transition to an anti-inflammatory period in which the risk of translocated intestinal flora and the subsequent development of infection increases.



Vocab:

- * *“Interstitial Pancreatitis No Necrosis, No Fluid Collections*
- * *“Exudative Pancreatitis No Necrosis, Yes Fluid Collections*
- * *“Necrotizing Pancreatitis”: Yes Necrosis, Usually Fluid Collections*
- * *“Acute Peripancreatic Fluid Collection”: <4 weeks, no necrosis*
- * *“Pseudocyst”: >4 weeks Encapsulated, no necrosis*
- * *“Acute Necrotic Collection” (ANCs): < 4weeks + Necrosis*
- * *“Walled Off Necrosis” (WON): >4weeks, Encapsulated + Necrosis*
- * *“Intraparenchymal Fluid Collections ” - Not a used term any more , per Revised Atlanta Criteria 2012. Instead use ANCs or WONs - NOT pseudocyst.*

Vascular Complications:

- * Splenic Vein and Portal Vein Thrombosis
 - o *Isolated gastric varices can be seen secondary to splenic vein occlusion*
- * Pseudo-aneurysm of the GDA and Splenic Arteries

Non-Vascular Complications:

- * Abscess, Infection, etc... as discussed
- * Gas, as a characteristic sign of an infected fluid collection, is detected in only 20% of cases of pancreatic abscesses.

Random Imaging Pearl:

- * On Ultrasound, an inflamed pancreas will be *hypoechoic* (edematous) when compared to the liver (opposite of normal).

Chronic pancreatitis (CP) represents the end result of prolonged inflammatory change leading to irreversible fibrosis of the gland. Acute pancreatitis and chronic pancreatitis are thought of as different disease processes, and most cases of acute pancreatitis do not result in chronic disease. So, acute doesn't have to lead to chronic (and usually doesn't), but chronic can still have recurrent acute.

Etiology: Same as acute pancreatitis, the most common causes are chronic alcohol abuse and cholelithiasis which together result in about 90% of the cases.

Imaging Findings: Findings can be thought of as early or late:

Early:

- * Loss of T1 signal (*pancreas is normally the brightest T1 structure in the body*)
- * Delayed Enhancement
- * Dilated Side Branches

Late:

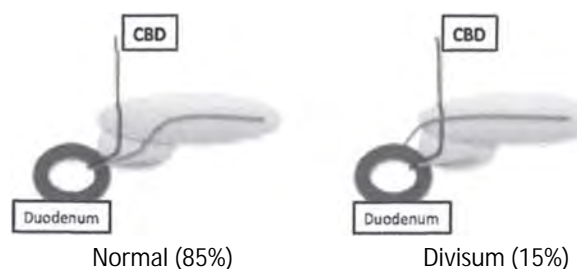
- * Commonly small, uniformly atrophic - but can have focal enlargement
- * Pseudocyst formation (30%)
- * Dilation and beading of the pancreatic duct with calcifications **** most characteristic finding of CP.**

<i>This vs That: Chronic Pancreatitis Duct Dilation vs Pancreatic Malignancy Duct Dilation</i>	
CP	Cancer
Dilation is Irregular	Dilation is uniform (<i>usually</i>)
Duct is < 50% of the AP gland diameter	Duct is > 50% of the AP gland diameter (<i>obstructive atrophy</i>)

Complications: Pancreatic cancer (*20 years of CP = 6% risk of Cancer*) is the most crucial complication in CP and is the biggest diagnostic challenge because focal enlargement of the gland induced by a fibrotic inflammatory pseudotumor may be indistinguishable from pancreatic carcinoma.

Pancreatic Divisum -*Anatomy Refresher:* There are two ducts, a major (Wirsung), and a minor (Santorini). The way I remember this is that “Santorini is Superior”, and “W” is in the back of the alphabet. “Santorini is also Small”, i.e. the minor duct.

Pancreatic Divisum is the most common anatomic variant of the human pancreas, and occurs when the main portion of the pancreas is drained by the minor or accessory papilla. The clinical relevance is an increased risk of Pancreatitis.



Uncommon Types and Causes of Pancreatitis				
Autoimmune Pancreatitis	Associated with elevated IgG4	Absence of Attack Symptoms	Responds to steroids	Sausage Shaped Pancreas, capsule like delayed rim enhancement around gland (like a scar)
Groove Pancreatitis	Duodenal and biliary obstruction, symptoms overlap with pancreatic cancer		Duodenal stenosis and /or strictures of the CBD in 50% of the cases	Soft tissue within the pancreaticoduodenal groove, with or without delayed enhancement
Tropic Pancreatitis	Young Age at onset, associated with malnutrition	Increased risk of adenocarcinoma		Multiple large calculi within a dilated pancreatic duct
Hereditary Pancreatitis	Young Age at Onset	Increased risk of adenocarcinoma	SPINK-1 gene	Similar to Tropic Pancreatitis
Ascaris Induced	Most commonly implicated parasite in pancreatitis			Worm may be seen within the bile ducts

When I Say - Auto Immune Pancreatitis	
I Say Auto Immune Pancreatitis	You Say IgG4
I Say IgG4	Autoimmune Pancreatitis Retroperitoneal Fibrosis Sclerosing Cholangitis Inflammatory Pseudotumor Riedel's Thyroiditis
This vs That: Auto Immune Pancreatitis vs Chronic Pancreatitis	
Auto Immune Pancreatitis	Chronic Pancreatitis
No ductal dilation	Ductal Dilation
No calcifications	Ductal Calcifications

Cystic Pancreatic Lesions

Pseudocyst: When you see a cystic lesion in the pancreas by far the *most common cause is going to be an inflammatory pseudocyst*, either from acute pancreatitis or chronic pancreatitis.

Simple Cysts: True epithelial lined cysts are rare, and tend to occur with syndromes such as VHL, Polycystic Kidney Disease, and Cystic Fibrosis.

Serous Cystadenoma (Grandma): The former term “microcystic adenoma” helps me think of a little old lady, which is appropriate for a lesion primarily found in elderly ladies. The lesion is benign, and classically described as heterogeneous mixed-density lesion made up of multiple small cysts, which resembles a sponge. They are more commonly (70%) located in the pancreatic head (*mucinous is almost always in the body or tail*). An additional key distinction is that it does NOT communicate with the pancreatic duct (*IPMNs do*). About 20% of the time they will have the classic central scar, with or without central calcifications (*mucinous calcifications are peripheral*).

Rarely, they can be unilocular. When you see a unilocular cyst with a lobulated contour located in the head of the pancreas, you should think about this more rare unilocular macrocystic serous cystadenoma subtype.

Mucinous Cystic Neoplasm (Mother): This pre-malignant lesion is “always” found in women, usually in their 50s. All are considered pre-malignant and need to come out. They are found in the body and tail (*serous was more common in the head*). There is generally no communication with the pancreatic duct (*IPMNs will communicate*). Peripheral calcifications are seen in about 25% of cases (*serous was more central*). They are typically unilocular. When multilocular, individual cystic spaces tend to be larger than 2cm in diameter (*serous spaces are typically smaller than 2cm*).

IPMN - Intraductal Papillary Mucinous Neoplasm: These guys are mucin-producing tumors that arise from the duct epithelium. They can be either side branch, main branch, or both.

Side Branch:

- o Typically appear as a small cystic mass, often in the head or uncinate process
- o If large amounts of mucin are produced it may result in main duct enlargement
- o Lesions less than 3cm, as usually benign

Main Branch:

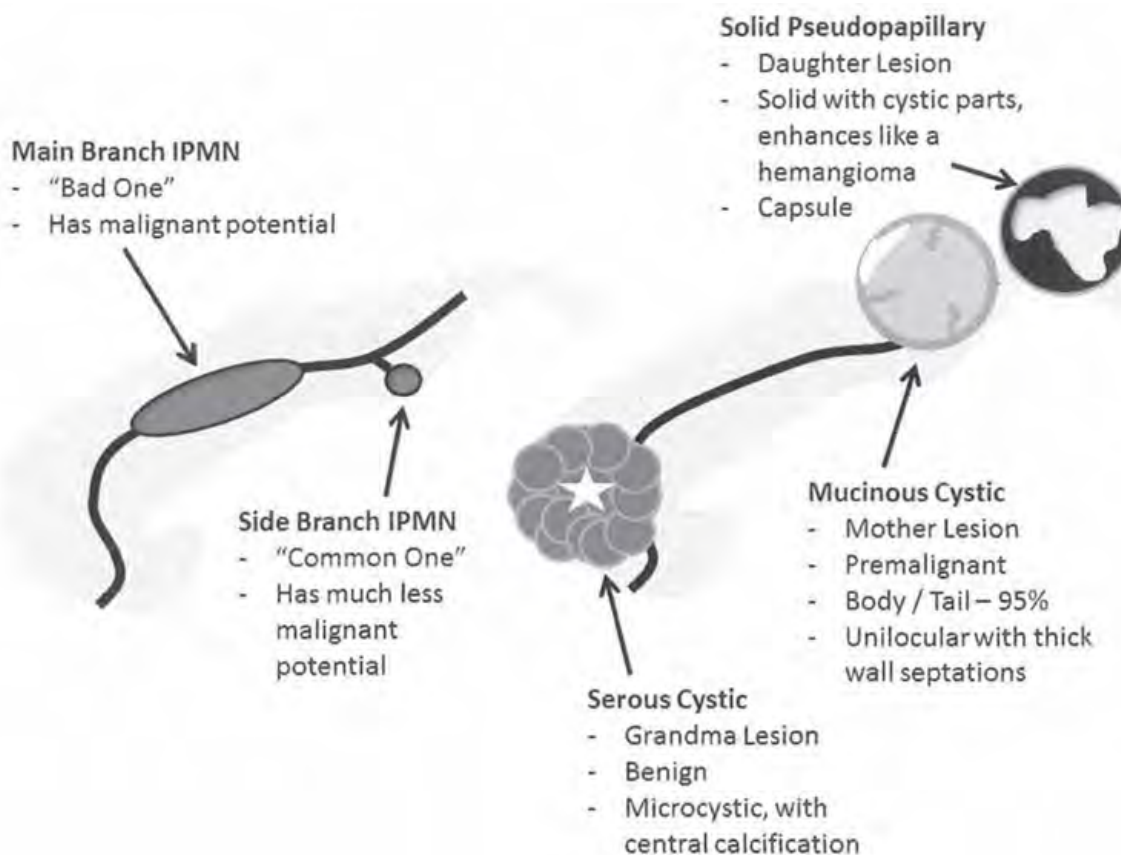
- o Produces diffuse dilation of the main duct
- o Atrophy of the gland and dystrophic calcifications may be seen - *mimicking Chronic Pancreatitis*
- o **Have a much higher % of malignancy compared to side branch**
- o All Main Ducts are considered malignant, and resection should be considered

Features Concerning For Malignancy:

- o Main duct >10mm (some sources say 1.5cm)
- o Diffuse or multifocal involvement
- o Enhancing nodules
- o Solid hypovascular mass

Solid Pseudopapillary Tumor of the Pancreas - (Daughter): Very rare, low grade malignant tumor that occurs almost exclusively in young (30s) females (usually Asian or Black). It is typically large at presentation, has a predilection for the tail, and has a “thick capsule.” Similar to a hemangioma it may demonstrate progressive fill in of the solid portions.

Cystic Pancreatic Lesion Summary:



Solid Pancreatic Lesions

Pancreatic Cancer basically comes in two flavors. (1) Ductal Adenocarcinoma - *which is hypovascular* and (2) Islet Cell / Neuroendocrine *which is hypervascular*.

Ductal Adenocarcinoma: In the setting of a multiple choice test, the finding of an enlarged gallbladder with painless jaundice is highly suspicious for pancreatic adenocarcinoma, especially when combined with migratory thrombophlebitis (*Trousseau's syndrome*). The peak incidence is in the 7th or 8th decade. The strongest risk factor is smoking.

Approximately, two thirds of these cancers arise from the pancreatic head. On ultrasound, obstruction of both the common bile duct, and the pancreatic duct is referred to as the "double duct sign". On CT, the findings are typically a hypovascular mass which is poorly demarcated and low attenuation compared to the more brightly enhancing background parenchyma.

The key to staging is assessment of the SMA and celiac axis, which if involved make the patient's cancer unresectable. Involvement of the GDA is ok, because it comes out with the whipple.

Additional Trivia Points about Pancreatic Adenocarcinoma:

- * Tumor Marker = C A 19-9
- * Hereditary Syndromes with Pancreatic CA:
 - o HNPCC, BRCA Mutation, Ataxia-Telangiectasia, Peutz-Jeghers
- * Small Bowel Follow Through: Reverse impression on the duodenum "Frostburg's Inverted 3 Sign" or a "Wide Duodenal Sweep." *The ABR would have to actually find a case of the inverted 3 to show it, but could ask it in words. The " Wide Duodenal Sweep " Could actually be shown.*

Periampullary Tumor: Defined as originating within 2cm of the major papilla. It can be difficult to differentiate from a conventional pancreatic adenocarcinoma as both obstruct the bile duct, and present as a mass in the pancreatic head. Basically, all you need to know about them is they can try and treat them with a Whipple and they have a better prognosis than pancreatic adenocarcinoma.

Islet Cell / Neuroendocrine: Neuroendocrine tumors are uncommon tumors of the pancreas. Typically hypervascular, with brisk enhancement during arterial or pancreatic phase. They can be thought of as non-functional or functional, and then subsequently further divided based on the hormone they make. They can be associated with both MEN 1, and Von Hippel Lindau.



Insulinoma: The most common type (about 75%). They are almost always benign (90%), solitary, and small (<2cm).

Islet Cell Tumor
- Enhancing in pancreatic tail

Gastrinoma: The second most common type overall, but most common type associated with MEN. They are malignant like 30-60%. They can cause increased gastric acid output and ulcer formation - Zollinger-Ellison syndrome.

The buzzword is Jejunal Ulcer = Zollinger Ellison.

Non-Functional: The 3rd most common type, usually malignant (80%), and are usually large and metastatic at the time of diagnosis.

Pediatric Pancreatic Tumor Considerations:

Age 1: Pancreatoblastoma

Age 6: Adenocarcinoma

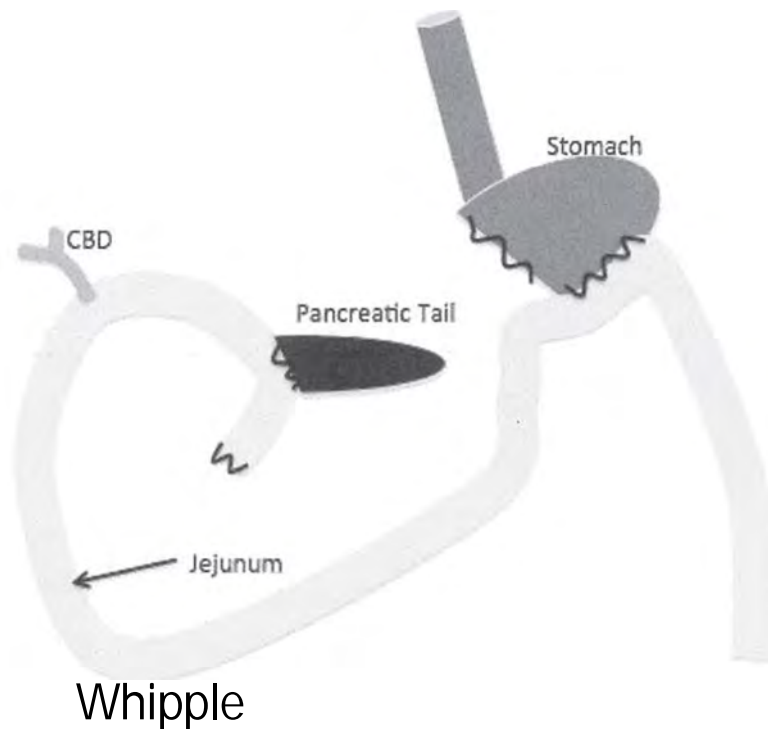
Age 15: Solid Pseudopapillary Tumor of the Pancreas

Surgical Correlates:

The Whipple Procedure:

The standard Whipple procedure involves resection of the pancreatic head, duodenum, gastric antrum, and almost always the gallbladder. A jejunal loop is brought up to the right upper quadrant for gastrojejunal, choledochojejunal or hepaticojejunal, and pancreatojejunal anastomosis.

An alternative method used by some surgeons is to perform a pancreatoduodenectomy to preserve the pylorus when possible. However, there is debate in the surgery literature with regard to which method should be the standard. In this pylorus-preserving pancreatoduodenectomy, the stomach is left intact and the proximal duodenum is used for a duodenojejunal anastomosis



Complications:

Delayed gastric emptying (*need for NG tube longer than 1- day*) and pancreatic fistula (*amylase through the surgical drain >50ml for longer than 7-10 days*), are both clinical diagnoses and are the most common complications after pancreatoduodenectomy. Wound infection is the third most common complication, occurring in 5%-20% of patients.

Transplant:

Pancreas transplant (usually with a renal transplant) is an established therapy for severe type 1 diabetes - which is often complicated by renal failure. The vascular anatomy regarding this transplant is quite complicated and beyond the scope of this text. Just know that the pancreas transplant receives arterial inflow from two sources: the donor SMA, *{which supplies the head via the inferior pancreaticoduodenal artery}* and the donor splenic artery, *{which supplies the body and tail}*. The venous drainage is via both the donor portal vein and the recipient SMV. Exocrine drainage is via the bowel *{in older transplant via the bladder}*.

The number one cause of graft failure is acute rejection. The number two cause of graft failure is splenic vein thrombosis. Splenic vein thrombosis usually occurs within the first 6 weeks of transplant. Venous thrombosis is much more common than arterial thrombosis in the pancreas, especially when compared to other transplants because the vessels are smaller and the clot frequently forms and propagates from the tied off stump vessels.

Both venous thrombosis and acute rejection can appear as reversed diastolic flow. Arterial thrombosis is also less of a problem because of the dual supply to the pancreas (via the Y graft). A point of trivia is that the resistive indices are not of value in the pancreas, because the organ lacks a capsule. The graft is also susceptible to pancreatitis, which is common < 4 weeks after transplant and usually mild. Increased rates of pancreatitis were seen with the older bladder drained subtype. “Shrinking Transplant” is a buzzword for chronic rejection, where the graft progressively gets smaller in size.

The Spleen

Normal Trivia

By the age of 15 the spleen reaches its normal adult size. The spleen contains both “red pulp” and “white pulp” which contribute to its tiger striped appearance during arterial phase imaging. The red pulp is filled with blood (a lot of blood), and can contain up to one liter of blood at any time. The spleen is usually about 20 HU more dense than the liver, and slightly more echogenic than the liver (equal to the left kidney). The splenic artery (which usually arises from the celiac trunk) is essentially an end vessel, with minimal collaterals. Occlusion of the splenic artery will therefore result in infarct of the spleen.

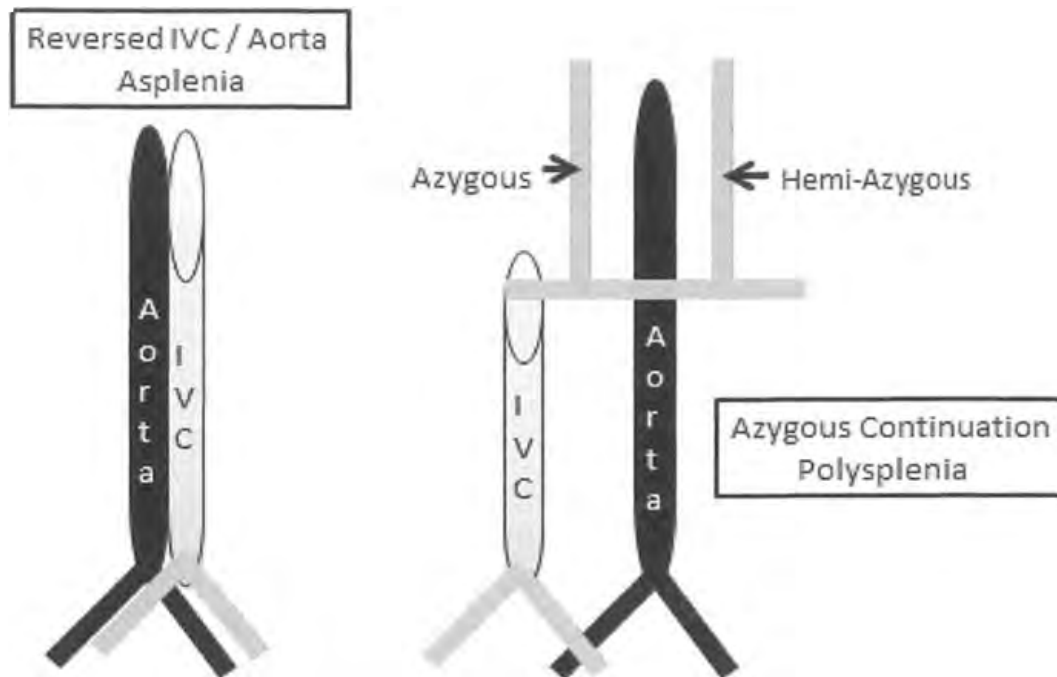
Pathology involving the spleen can be categorized as either congenital, acquired (as the sequella of trauma or portal hypertension), or related to a “mass.” A general rule is that most things in the spleen are benign with exception of lymphoma or the rare primary angiosarcoma.

Heterotaxia Syndromes

These lend themselves well to multiple choice test questions and are probably the highest yield topic to understand regarding the spleen. The major game played on written tests is “left side vs right side.” So what the hell does that mean? I like to start in the lungs. The right side has two fissures (major and minor). The left side has just one fissure. So if I show you a CXR with two fissures on each side, (a left sided minor fissure), then the patient has two right sides. Thus the term “bilateral right sidedness.” Well what else is a right sided structure? The liver. So, these patients won’t have a spleen (the spleen is a left sided structure). The opposite is true, bilateral left sided patients have polysplenia.

Key trivia to know:

Right Sided	Left Sided
Two Fissures in Left Lung	One Fissure in Right Lung
Asplenia	Polysplenia
Cardiac Malformations	Biliary Atresia
Reversed Aorta / IVC	Azygous Continuation of IVC



Accessory Spleens These are very common, we see them all the time. Some random trivia that might be testable includes the fact that sulfur colloid could be used to differentiate a splenule from an enlarged pathologic lymph node. Additionally, in the scenario where a patient is post splenectomy for something like ITP or autoimmune hemolytic anemia, an accessory spleen could hypertrophy and present as a mass. Hypertrophy of an accessory spleen can also result in a recurrence of the original hematologic disease process.

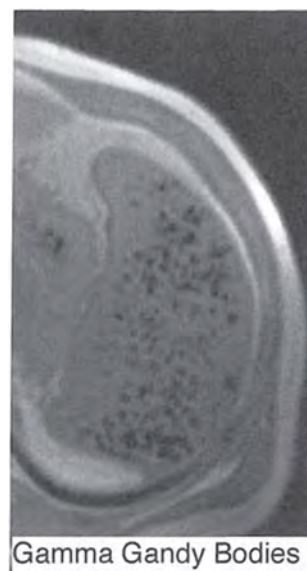
Wandering Spleen A normal spleen that “wanders” off and is in an unexpected location. Because of the laxity in the peritoneal ligaments holding the spleen, a wandering spleen is associated with abnormalities of intestinal rotation. The other key piece of trivia is that unusual locations set the spleen up for torsion and subsequent infarction. A chronic partial torsion can actually lead to splenomegaly or gastric varices.

Trauma The spleen is the most common solid organ injured in trauma. This combined with the fact that the spleen contains a unit or so of blood means splenic trauma can be life threatening.

Splensosis: This occurs post trauma where a smashed spleen implants and then recruits blood supply. The implants are usually multiple and grow into spherical nodules typically in the peritoneal cavity of the upper abdomen (*but can be anywhere*). It's more common than you think and has been reported in 40-60% of trauma. Again, Tc Sulfur colloid can confirm that the implants are spleen and not ovarian mets or some other more serious pathology.

Gamma Gandy Bodies (Siderotic Nodule): These are small foci of hemorrhage in the splenic parenchyma that are usually associated with portal hypertension. They are T2 dark.

Gradient is the most sensitive sequence.



Sarcoidosis: Sarcoid is a disease of unknown etiology that results in noncaseating granulomas and forms in various tissues of the body (*complete discussion in the chest section of this text*). The spleen is involved in 50% - 80% of patients. Splenomegaly is usually the only sign. However, aggregates of granulomatous splenic tissue in some patients may appear on CT as numerous discrete 1-2cm hypodense nodules. Rarely, it can cause a massive splenomegaly and possibly rupture. Don't forget that the gastric antrum is the most common site in the GI tract.

Peliosis: This is a rare condition characterized by multiple blood filled cyst-like spaces in a solid organ (*usually the liver - peliosis hepatitis*). When you see it in the spleen it is usually also with the liver (isolated spleen is extremely rare). The etiology is not known, but for the purpose of multiple choice tests it occurs in women of OCPs, men on anabolic steroids, **people with AIDS, renal transplant patients** (up to 20%), and Hodgkin lymphoma. It's usually asymptomatic but can explode spontaneously.

Splenic Vascular Abnormalities

Splenic artery' aneurysm is the most common visceral arterial aneurysm. Pseudoaneurysm can occur in the setting of trauma and pancreatitis. The incidence is higher in women of child bearing age who have had two or more pregnancies (*4x more likely to get them, 3x more likely to rupture*). It's usually sacular and in the mid to distal artery. They usually fix them when they get around 2-3cm. Major "F" up to avoid: Don't call them a hypervascular pancreatic islet cell mass and biopsy them.

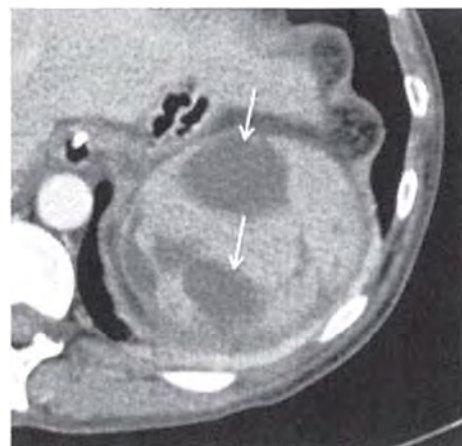
Splenic vein thrombosis frequently occurs as the result of pancreatitis. Can also occur in the setting of diverticulitis or Crohns. Can lead to isolated gastric varices.

Infarction can occur from a number of conditions. On a multiple choice test the answer is sickle cell. The imaging features are classically a wedge-shaped, peripheral, low attenuation defect.

Splenic Infections

Most common radiologically detected splenic infection is histoplasmosis (with multiple round calcifications). Splenic TB can have a similar appearance (but much less common in the US). Another possible cause of calcified granuloma in the spleen in brucellosis, but these are usually solitary and 2cm or larger. They may have a low density center, encircled by calcification giving the lesion a "bull's eye" appearance.

In the immunocompetent patient, **splenic abscess** is usually due to an aerobic organism. **Salmonella is the classic bug** - which develops in the setting of underlying splenic damage (trauma, or sickle cell). In immunocompromised patient's, unusual organisms such as fungi, TB, MAI, and PCP can occur and usually present as multiple micro-abscesses. Occasionally, fungal infections may show a "bulls-eye" appearance on ultrasound.



Multiple Splenic Abscesses

Splenic Size - Too Big vs Too Small:

It's good to have a differential for a big spleen and a small spleen.

Small Spleen	Big Spleen
Sickle Cell	Passive Congestions (heart failure, portal HTN, splenic vein thrombosis)
Post Radiation	Lymphoma
Post Thorotrast	Leukemia
Malabsorption Syndromes (ulcerative colitis > crohns)	Gauchers



Feltz's Syndrome - abnormality of granulocytes, with a triad of: **(1)**

Splenomegaly, (2) Rheumatoid Arthritis, (3) Neutropenia

Benign Masses of the Spleen

Cysts:

Post traumatic cysts (pseudocysts) are the most common cystic lesion in the spleen. They can occur secondary to infarction, infection, hemorrhage or extension from a pancreatic pseudocyst. As a point of trivia they are “pseudo” cysts because they have no epithelial lining. They may have a thick wall or prominent calcifications peripherally.

Epidermoid cysts are the second most common cystic lesion in the spleen. They are congenital in origin. As a point of absolutely worthless trivia, they are “true” cysts and have an epithelial lining. They typically grow slowly and are usually around 10cm at the time of discovery. They can cause symptoms if they are large enough. They are solitary 80% of the time, and have peripheral calcifications 25% of the time.

Hydatid or Echinococcal cysts are the third most common cystic lesion in the spleen (most common worldwide). They are caused by the parasite *Echinococcus Granulosus*. Hydatid cysts consist of a spherical “mother cyst” that usually contains smaller “daughter cysts.” Internal septations and debris are often referred to as “hydatid sand.” Another sign described is the “water lily sign.” The “water lily sign” is seen when there is detachment of the endocyst membrane resulting in floating membranes within the pericysts (looks like a water lily). This was classically described on CXR in pulmonary echinococcal disease.

Hemangioma is the most common benign neoplasm in the spleen. This dude is usually smooth and well margined demonstrating contrast uptake and delayed washout. *The classic peripheral nodular discontinuous enhancement seen in hepatic lesions may not occur*, especially if the tumor is smaller than 2cm.

Lymphangiomas are rare entities in the spleen but can occur. Most occur in childhood. They may be solitary or multiple, although most occur in a subcapsular location. Diffuse lymphangiomas may occur (lymphangiomatosis).

Hamartomas are also rare in the spleen, but can occur. Typically this is an incidental finding. Most are hypodense or isodense and show moderate heterogeneous enhancement. They can be hyperdense if there is hemosiderin deposition.



Littoral Cell Angioma is a zebra that shows up occasionally in books and possibly on multiple choice tests. Clinical hypersplenism is almost always present. Usually presents as multiple small foci which are hypoattenuating on late portal phase. MR shows hemosiderin (low T1 and T2).

Malignant Masses of the Spleen

Most things that occur in the spleen are benign. Other than lymphoma (discussed below) it is highly unlikely that you will encounter a primary malignancy of the spleen (*but if you do it's likely to be vascular*). For the purposes of academic discussion (and possible multiple choice trivia) angiosarcoma is the most common. It is aggressive and has a poor prognosis. On CT it can manifest as a poorly defined area of heterogeneity or low density in an enlarged spleen. They can contain necrosis and get big enough to rupture. Contrast enhancement is usually poor. Yes, these can occur from prior thorotrast exposure.

Lymphoma is the most common malignant tumor of the spleen, and is usually seen as a manifestation of systemic disease. *Splenomegaly is the most common finding* (and maybe the only finding in low grade disease). Although both Hodgkins and Non-Hodgkins types can involve the spleen, Hodgkins type and high grade lymphomas can show discrete nodules of tumor. With regard to imaging, they are low density on CT, T1 dark, and are PET hot.

Metastatic disease to the spleen is rare. When it does occur, it occurs via common things (Breast, Lung).

3

Urinary

Prometheus Lionhart, M.D.

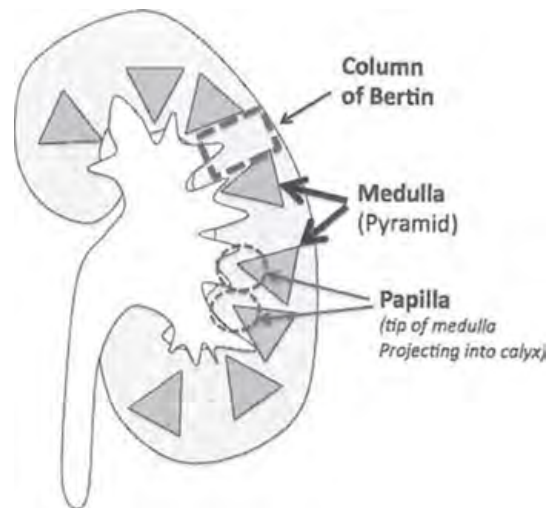


Renal pathology can easily be shown on US, CT, or MRI - but because so many things look alike, associations and trivia should be more heavily weighted.

High Yield Topics:

- Renal Cell Carcinoma Subtypes, Staging, and Associations
- Congenital Renal
- Bladder Cancer Subtypes
- Renal Doppler

Normal Anatomy: The normal adult kidney is shaped like a bean, with a smooth (often lobulated) outer border. The kidney is surrounded by a thick capsule outlined by echogenic perirenal fat. This echogenic fat is contiguous with the renal sinus, filling the middle of the kidney. The cortex extends centrally into the middle of the kidney, separated by slightly less echogenic medullary pyramids. The normal kidney should be between 9cm and 15cm in length.



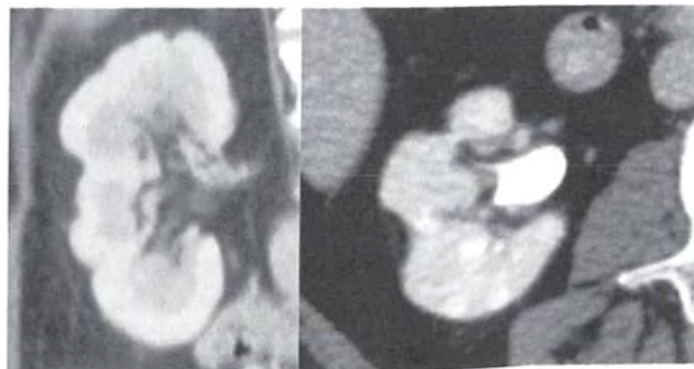
The echogenicity of the kidney should be equal to or slightly less than the liver and spleen. If the renal echogenicity is greater than the liver, this indicates some impaired renal function (medical renal disease). Liver echogenicity greater than the kidney indicates a fatty liver.

Variant Anatomy

Fetal Lobulation - The fetal kidneys are subdivided into lobes that are separated with grooves. Sometimes this lobulation persists into adult life. The question is always;

Fetal Lobulation vs Scarring:

- * **Lobulation** = renal surface indentations overlie the space between the pyramids
- * **Scarring** = renal surface indentations overlie the medullary pyramids.

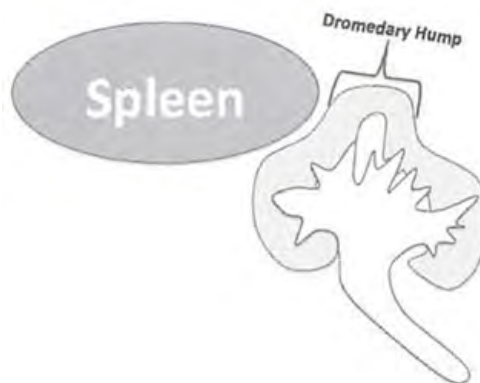


Fetal Lobulation

Renal Scarring

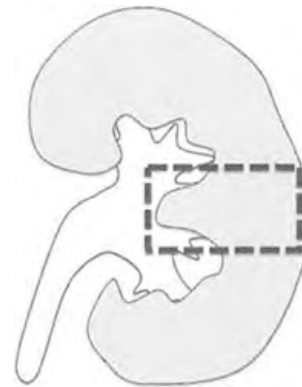
-Indentations Between Pyramids -Indentations Over Pyramids

Dromedary Hump - Focal bulge on the left kidney, which forms as the result of adaptation to the adjacent spleen.



Prominent (or hypertrophied) Column of Bertin -

Normal variant in which hypertrophied cortical tissue located between the pyramids results in splaying of the sinus. Other than the hypertrophy it looks totally normal. It will enhance the same as adjacent parenchyma.



Prominent Column of Bertin

Renal Agenesis - Congenital absence of one or both kidneys. If it's unilateral this can be asymptomatic. If it's bilateral think about the "Potter Sequence." When it's unilateral (it's usually sporadic), but for the purpose of multiple choice think about associated GYN anomalies in women (70% of women with unilateral renal agenesis have associated genital anomalies - **unicornuate uterus**). With regard to men, **20% with renal agenesis have absence of the ipsilateral epididymis and vas deferens or an ipsilateral seminal vesicle cyst.**

Associations: Ipsilateral seminal vesicle cysts, absent ipsilateral ureter, absent ipsilateral hemitrigone and absent ipsilateral vas deferens

- o **Potter Sequence:** Insult (maybe ACE inhibitors) = kidneys don't form, if kidneys don't form you can't make piss, if you can't make piss you can't develop lungs (pulmonary hypoplasia).
- o **Mayer-Rokitansky-Kuster-Hauser.** - Mullerian duct anomalies including absence or atresia of the uterus. Associated with unilateral renal agenesis.
- O Lying Down Adrenal or "Pancake Adrenal" Sign** - describes the elongated appearance of the adrenal not normally molded by the adjacent kidney. It can be used to differentiate surgical absent vs congenital absent.

Horseshoe Kidney - This is the most common fusion anomaly. The IMA gets hung up on the IMA. Complications include Traumatic Injury (gets crushed against vertebral body), UPJ Obstruction, Recurrent Infection, Recurrent Stones, Wilms Tumor (8x higher), TCC (from all those infections). A rare situation, but known association is the renal carcinoid occurring in horseshoe kidney. Turners syndrome is a classically tested association.

Crossed Fused Renal Ectopia - One kidney comes across the midline and fuses with the other. "The Ectopic Kidney is Inferior." The left kidney more commonly crosses. Complications include stones, infection, and hydronephrosis (50%).

Renal Masses

Renal Cell Carcinoma:

The most common primary renal malignancy. RCC till proven otherwise: (a) Enhances with contrast (> 15 H.U.), (b) calcifications in a fatty mass. Risk factors include tobacco use, syndromes like VHL, chronic dialysis (> 3 years), family history. These dudes make hypervascular mets. They are ALWAYS lytic when they met to the bones.

- * **Pseudoenhancement:** A less than 10 HU increase in attenuation is considered within the technical limits of the study and is not considered to represent enhancement. More rare once a cyst is larger than 1.5cm.

Subtypes:

- **Clear Cell** - Most common subtype. This is the one that is associated with VHL. It is typically more aggressive than papillary, and will enhance equal to the cortex on corticomedullary phase.
- # **Papillary** - This is the second most common type. It is usually less aggressive than clear cell (more rare subtypes can be very aggressive). They are less vascular and will not enhance equal to the cortex on corticomedullary phase. They also are in the classic T2 dark differential (along with lipid poor AML, and hemorrhagic cyst).
- * **Medullary** - Associated with Sickle Cell Trait. It's highly aggressive, and usually large and already metastasized at the time of diagnosis.
- * **Chromophobe** - All you need to know is that it's associated with Birt Hogg Dube.

Conventional RCC Staging:

Stage 1: Limited to Kidney and < 7 cm

Stage 2: Limited to Kidney but > 7 cm

Stage 3: Still inside Gerota's Fascia

A: Renal Vein Invaded

B: IVC below diaphragm

C: IVC above diaphragm

Stage 4: Beyond Gerota's Fascia

Ipsilateral Adrenal

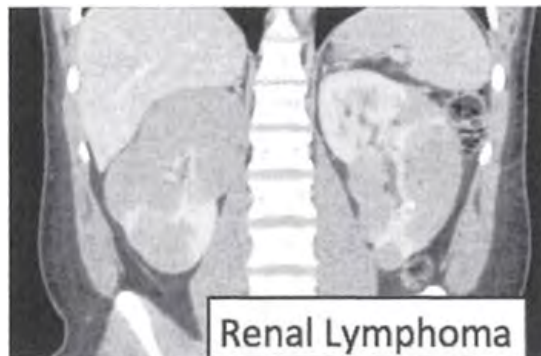
Subtype	Syndrome / Association
Clear Cell	Von Hippel-Lindau
Papillary	Hereditary papillary renal carcinoma
Chromophobe	Birt Hogg Dube
Medullary	Sickle Cell Trait

Sneaky Move

Does Adult Polycystic Kidney Disease increase your risk for RCC???? - Well No, but sorta. The genetic syndrome does NOT intrinsically increase your risk. However, dialysis does. Who gets dialysis?? People with APCKD. It would be such a crap way to ask a question - but could happen. If you are asked, I'm recommending you say no to the increased risk, - unless the question writer specifies that the patient is on dialysis.

Renal Masses That Are Not RCC

Renal Lymphoma - This can literally look like anything. Having said that the most common appearance is bilateral (it is usually bilateral) enlarged kidneys, with small, low attenuation cortically based solid nodules or masses. A solitary mass is seen in about 1/4 of the cases.



Renal Leukemia: The kidney is the most common visceral organ involved. Typically the kidneys are smooth and enlarged. Hypodense lesions are cortically based only, with little if any involvement of the medulla.

Oncocytoma This is the second most common benign tumor (after AML). It looks a lot like a RCC. A **central scar** is present 33% of the time. There will be no malignant features (such as vessel infiltration). A syndrome associated with bilateral oncocytomas is Birt Hogg Dube (they also get chromophobe RCC). They cannot be distinguished from RCC on imaging and must be treated as RCC till proven otherwise.

If they want to ask about an Oncocytoma they can show it 3 ways: (1) Solid Mass with central scar - CT or MRI, (2) On Ultrasound “spoke wheel” vascular pattern, (3) on PET CT it will be hotter than surrounding renal cortex.



The PET Trick:

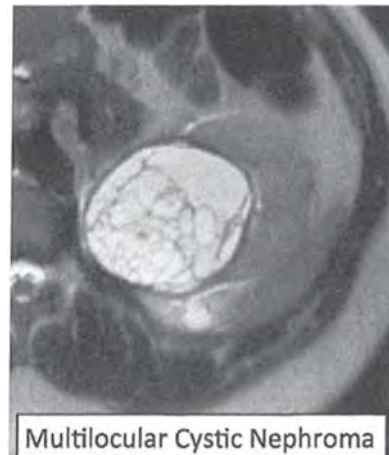
RCC is typically COLDER than surrounding renal parenchyma on PET,

Oncocytoma is typically HOTTER than surrounding renal parenchyma on PET,

Angiomyolipoma (AML)- This is the most common benign tumor of the kidney. Almost always (95%) of them have macroscopic fat, and this is the defining feature. They are usually incidental (in the real world). The things to know about them are (1) They are associated with Tubular Sclerosis (2) They can bleed if they get big enough (>4cm). It's controversial if they grow or bleed more in pregnancy (if they ask you, I guess you should say yes - because that's the old knowledge but some modern papers are saying not for sure). They should never have calcifications (that's probably a RCC). They can be lipid poor (about 5% are), and those are T2 dark.

Multilocular Cystic Nephroma - "Non-communicating, fluid-filled locules, surrounded by thick fibrous capsule." By definition these things are characterized by the absence of a solid component or necrosis. **Buzzword is "protrudes into the renal pelvis."** The question is likely the bimodal occurrence (4 year old boys, and 40 year old women).

I like to think of this as the *Michael Jackson lesion* - it loves young boys and middle aged women.



Cystic Disease

Bosniak Cyst Classification:

- * Class 1: Simple - less than 15 H.U. with no enhancement
- * Class 2: Hyperdense (< 3cm). Thin calcifications, Thin septations
- * Class 2F: Hyperdense (>3cm). Minimally thickened calcifications (5% chance cancer)
- * Class 3: Thick Septations, Mural Nodule (50% chance cancer)
- * Class 4: Any enhancement (>15 H.U.)



Class 1:
- Simple Anechoic



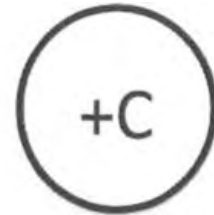
Class 2:
-Hyperdense (<3cm)
-Thin Calcifications
-Thin Septations



Class 2F:
-Hyperdense (>3cm)
-Thin Calcifications
-< 5% chance of CA



Class 3:
-Thick Calcifications
-Mural Nodule
- 50% chance of CA



Class 4:
-Any Enhancement

Hyperdense cysts: Basically, if the mass is greater than 70H.U. and homogenous it's benign (hemorrhagic or proteinaceous cyst) 99.9% of the time.

Classic DDX: The T2 Dark Cyst

- j - Hemorrhagic Cyst
- Lipid Poor AML
- Papillary Sub Type RCC

Autosomal Dominant Polycystic Kidney Disease (ADPKD) - Kidneys get progressively larger and lose function (you get dialysis by 5th decade). Hyperdense contents & calcified wall are frequently seen due to prior hemorrhage. What you need to know is: (1) it's Autosomal Dominant "ADult", (2) They get cysts in the liver 70% of the time, and (3) they get Berry Aneurysms. As mentioned before they don't have an intrinsic risk of cancer, but do get cancer once they are on dialysis.

Autosomal Recessive Polycystic Kidney Disease (ARPKD) - These guys get HTN, and renal failure. The liver involvement is different than the adult form. Instead of cysts they have abnormal bile ducts and fibrosis. This **congenital hepatic fibrosis is ALWAYS present in ARPKD**. The ratio of liver and kidney disease is inversely related. *The worse the liver is the better the kidneys do. The better the liver is the worse the kidneys are.* On ultrasound the kidneys are smoothly enlarged and diffusely echogenic, with a loss of corticomedullary differentiation.

Uremic Cystic Kidney Disease - About 40% of patients with end stage renal disease develop cysts. This rises with duration of dialysis in about 90% in patients after 5 year of dialysis. The thing to know is: **Increased risk of malignancy with dialysis.**

Von Hippel Lindau - Autosomal dominant multi-system disorder. 50-75% have renal cysts. 25-50% develop RCC (clear cell)

- * Pancreas: - Cysts, Serous Microcystic Adenomas, Neuroendocrine (islet cell) tumor
- * Adrenal: Pheochromocytoma (often multiple)
- * CNS: Hemangioblastoma of the cerebellum, brain stem, and spinal cord

Tuberous Sclerosis - Autosomal dominant multi-system disorder. You have hamartomas everywhere (brain, lung, heart, skin, kidneys). The renal findings are bilateral multiple angiomyolipomas. They also have renal cysts, and occasionally RCC (same rate as general population, but in younger patient population). With regard to other organ systems:

- * Lung - LAM - thin walled cysts and chylothorax
- * Cardiac - Rhabdomyosarcoma (typically involve cardiac septum)
- * Brain - Giant Cell Astrocytoma, Cortical and subcortical tubers, subependymal nodules
- * Renal - AMLs, RCC (in younger patients)

Lithium Nephropathy -Occurs in patients who take lithium long term. Can lead to diabetes insipidus and renal insufficiency. The kidneys are normal to small in volume with multiple (innumerable) tiny cysts, usually 2-5mm in diameters. These "microcysts" distinguishable from larger cysts associated with acquired cystic disease of uremia. They are probably going to show this on MRI with the history of bipolar disorder.

ADPKD	Cysts in Liver	Kidneys are BIG
VHL	Cysts in Pancreas	
Acquired (Uremic)		Kidneys are small

Multicystic Dysplastic Kidney This is a peds thing, where you have multiple tiny cysts forming in utero. What you need to know: (1) that there is “no functioning renal tissue,” (2) contralateral renal tract abnormalities occur like 50% of the time.

MCDK vs Bad Hydro?

- * *In hydronephrosis the cystic spaces are seen to communicate.*
- * *In difficult cases renal scintigraphy can be useful. MCDK will show no excretory function.*

Peripelvic Cyst vs Parapelvic Cyst-

- * *Peri: Originates from renal sinus, mimics hydro*
- * *Para: Originates from parenchyma, may compress the collecting system*

Renal Infection

Pyelonephritis - This is a clinical diagnosis. However since ED doctors are universally stupid, you do end up diagnosing it. It's associated with stones. The most common organism is E. Coli. In acute bacterial nephritis, alternating bands of hypo and hyperattenuation (striated nephrogram) are seen. These wedge shaped areas are related to decreased perfusion. Perinephric stranding is also commonly seen.

Striated Nephrogram DDx:

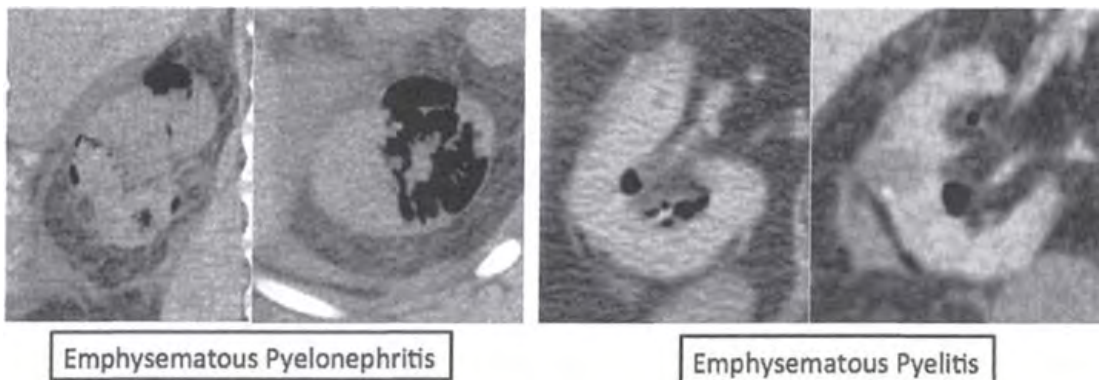
- Acute ureteral obstruction
- * Acute pyelonephritis
- Medullary sponge kidney
- Acute renal vein thrombosis
- * Radiation nephritis
- Acutely following renal contusion
- * Hypotension (bilateral)
- * Infantile polycystic kidney (bilateral)

Abscess - Pyelo may be complicated by abscess, which can present on CT as round or geographic low attenuation collections that do not enhance centrally, but do have an enhancing rim. Bigger than 3cm and these guys might visit the IR section for drainage.

Chronic Pyelonephritis - Sort of a controversial entity. It is not clear whether the condition is an active chronic infection, arises from multiple recurrent infections, or represents stable changes from a remote single infection. The imaging findings are characterized by renal scarring, atrophy and cortical thinning, with hypertrophy of residual normal tissue. Basically, you have a small deformed kidney, with a bunch of wedge defects, and some hypertrophied areas.

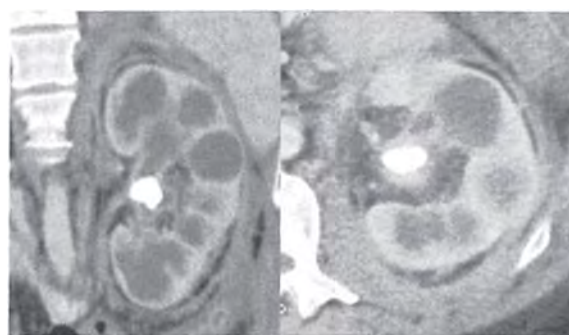
Emphysematous Pyelonephritis - This is a life threatening necrotizing infection characterized by gas formation within or surrounding the kidney. What you need to know (1) it's really bad, (2) diabetics almost exclusively get it, (3) echogenic foci with dirty shadowing on ultrasound,

Emphysematous Pyelitis - This is less bad relative to emphysematous pyelonephritis. The gas is localized to the collecting system. It's more common in women, diabetics, and people with urinary obstruction. Radiographic finding is gas outlining the ureters and dilated calices.



Pyonephrosis - An infected or obstructed collecting system (which is frequently enlarged). Can be from a variety of causes; stones, tumor, sloughed papilla secondary to pyelonephritis. Can totally jack your renal function if left untreated. Fluid-Fluid level in the collecting system can be seen on US. CT has trouble telling the difference between hydro and pyonephrosis.

Xanthogranulomatous Pyelonephritis (XGP) - chronic destructive granulomatous process that is basically always seen with a staghorn stone acting as a nidus for recurrent infection. You can have an associated psoas abscess with minimal perirenal infection. It's an Aunt Minnie, with a very characteristic "Bear Paw" appearance on CT. The kidney is not functional, and sometimes nephrectomy is done to treat it.



Xanthogranulomatous Pyelonephritis - *Bear Paw*

Papillary Necrosis: This is ischemic necrosis of the renal papillae, most commonly involving the medullary pyramids. **Diabetes is the most common cause.** Other important causes include; pyelonephritis (especially in kids), sickle cell, TB, analgesic use, and cirrhosis. The appearance of a necrotic cavity in the papillae with linear streaks of contrast inside the calyx has been called a “lobster claw sign.” Filling defects might be seen in the calyx

TB- The most common extrapulmonary site of infection is the urinary tract. There are features of papillary necrosis and parenchymal destruction. You can have extensive calcification. Basically you end up with a shrunken calcified kidney “putty kidney.” This end stage appearance is essentially an auto nephrectomy. The “Kerr Kink” is a sign of renal TB with scarring leading to a sharp kink at the pelvi-ureteric junction.

HIV Nephropathy - Normal sized (or **big**) **kidneys that are echogenic on US.** Loss of the renal sinus fat appearance has also been described (it’s edema in the fat, rather than loss of the actual fat).

Disseminated PCP in HIV patients can result in punctate (primarily cortical) calcifications.

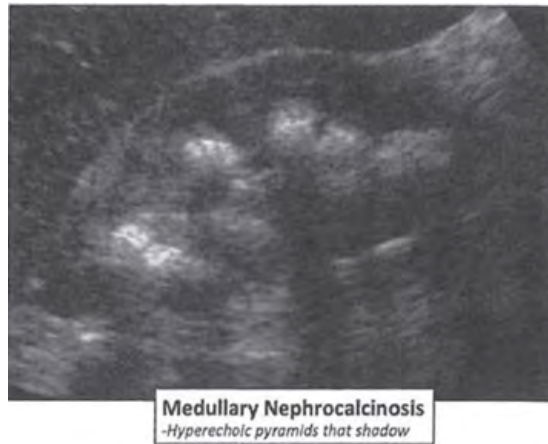
Renal Calcification /Misc:

Nephrolithiasis (kidney stones) - They are very common.

- * *Calcium Oxalate* - by far the most common type (75%)
- * *Struvite Stone* - More common in women, and associated with UTI
- * *Uric Acid* - “Unseen” on x-ray
- * *Cystine* - rare, and associated with congenital disorders of metabolism
- * *Indinavir* - Stones in HIV patients , which are **the ONLY stones not seen on CT.**

Cortical Nephrocalcinosis - This is typically the sequella of cortical necrosis, which can be seen with an acute drop in blood pressure (shock, post partum, bum patients, etc...). It starts out as a hypodense non-enhancing rim that later develops thin calcifications. Mimic is disseminated PCP.

Medullary Nephrocalcinosis - Hyperechoic renal papilla / pyramids which may or may not shadow. There are multiple causes, the most common of which is probably hyper PTH (*a few texts say medullary sponge kidney is most common*). Other things to think about include Lasix use (in a child), Renal Tubular Acidosis (distal subtype - or type 1), medullary sponge kidney (if asymmetric). RTA and Hyperparathyroid usually cause a more dense calcification than medullary sponge kidney.



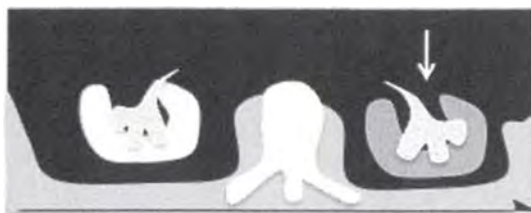
Medullary Sponge Kidney - A congenital cause of medullary nephrocalcinosis (usually asymmetric). The underlying mechanism is a cystic dilation of the collecting tubules of the kidney - so the association with Ehler Danlos makes sense. The association with Carolis sorta makes sense. The association with Beckwith-Weidman doesn't really make sense (and therefore is the most likely to be tested).

Think about medullary sponge kidney with unilateral medullary nephrocalcinosis.

This vs That: Delayed vs Persistent Nephrogram

Delayed Nephrogram - One kidney enhances and the other doesn't (or does to a lesser degree). Basically this is happening from pressure on the kidney (either extrinsic from a Page kidney situation), or intrinsic from an obstructing stone.

Persistent Nephrogram - This is seen with hypotension/shock, and ATN. They can show this several ways, one would be on a plain film of the abdomen (with dense kidneys), the second would be on CT. The tip offs are going to be that they tell you the time (3 hours etc...), and it's gonna be bilateral.



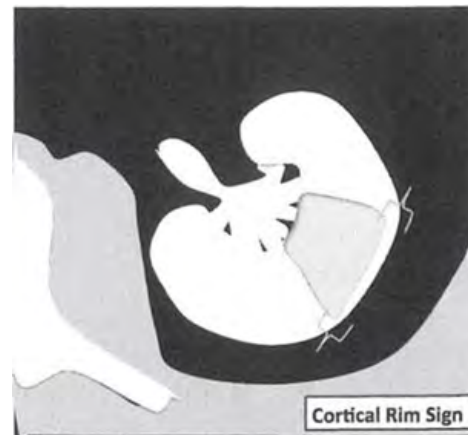
Delayed Nephrogram



Persistent Nephrogram

Page Kidney - Sequella of long standing compression of the kidney by some kind of subcapsular collection (hematoma or seroma). The classic scenario is post lithotripsy subcapsular hematoma, with follow up showing a shrunk up kidney.

Renal Infarct - So wedge shaped hypodensities in the kidney can be seen with lots of stuff (infarct, tumor, infection, etc...). Renal infarcts are most easily identified on post contrast imaging in the cortical phase. If the entire renal artery is out, well then it won't enhance (duh). Two tricks that they could pull are the (1) "Cortical Rim Sign" - which is absent immediately after the insult, but is seen 8 hours to days later. You have a dual blood supply, which allows the cortex to stay perfused. (2) "Flip Flop Enhancement" can be seen where a region of hypodensity / poor enhancement on early phases becomes relatively hyperdense on delayed imaging.



Renal Vein Thrombosis - Numerous causes including dehydration, indwelling umbilical venous catheters (most common in neonates), and nephrotic syndrome (most common in adults). This can mimic a renal stone; presenting with flank pain, an enlarged kidney, and a delayed nephrogram. On Doppler they are going to show you Reversed arterial diastolic flow, and absent venous flow. **This is discussed again below - in the transplant section.*

Renal Trauma

Obviously the kidney can get injured in trauma (seen in about 10%). Injury can be graded based on the presence of hematoma -> laceration -> involvement of the vein, artery, or UPJ obstruction. Delayed imaging may be helpful to demonstrate a urine leak.

Terminology:

- *"Fractured Kidney"* - Severe laceration, which extends the full length of the renal parenchyma.
- *"Shattered Kidney"* - A kidney with 3 or more fragments - representing the most severe form of renal fracture.

Renal Transplant

Renal transplant is the best treatment for end stage renal disease, and the quality of life is significantly better than that of a long term dialysis patient. The transplanted kidney is most commonly placed in the extraperitoneal iliac fossa so that the allograft can be anastomosed with the iliac vasculature and urinary bladder. You can think of complications in 3 main flavors (a) Urologic, (b) Vascular, (c) Cancer.

Normal: You should have a sharp systolic upstroke, with forward flow in diastole. **RI** should be < 0.8 .

Urologic Complications:

Urinoma - This is usually found in the first 2 weeks post op. Urine leak or urinoma will appear as an anechoic fluid collection with no septations, that is rapidly increasing in size. MAG 3 nuclear medicine scan can be used to demonstrate this (or the cheaper ultrasound).

Hematoma - Common immediately post op. Usually resolves spontaneously. Large hematoma can produce hydro. Acute hematoma will be echogenic, and this will progressively become less echogenic (with older hematomas more anechoic and septated).

Lymphocele - Lymphoceles typically occur **1-2 months after transplant**. They are caused by leakage of lymph from surgical disruption of lymphatics. The fluid collection is usually medial to the transplant (between the graft and the bladder).

Chronic Rejections - This occurs one year post transplant. The kidney may enlarge, and you can lose corticomedullary differentiation. The RIs will elevate (>0.7) which is nonspecific.

Calculous Disease - Compared with the general population, transplant kidneys are at increased risk of stone formation.

Vascular Complications:

Renal Artery Thrombosis - Occurs in 1% of renal transplants within the 1st week. Typically caused by kinking, hypercoagulation, or hyperacute rejection.

Renal Artery Stenosis - Occurs in 5-10% renal transplants. This usually occurs at the anastomosis. Criteria include:

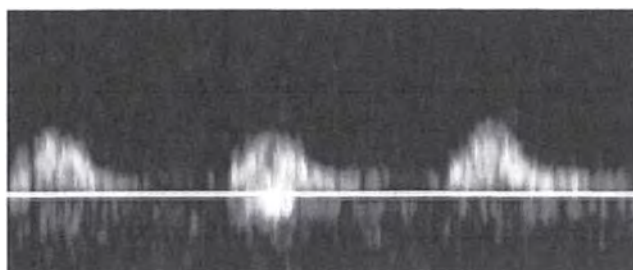
- PSV > 200-300 cm/s.
- PSV ratio >3.0 (External Iliac Artery /RA)
- * Tardus Parvus: Measured at the Main Renal Artery Hilum (NOT at the arcuates)
- * Anastomotic Jetting

What is this Tardus Parvus you speak of?

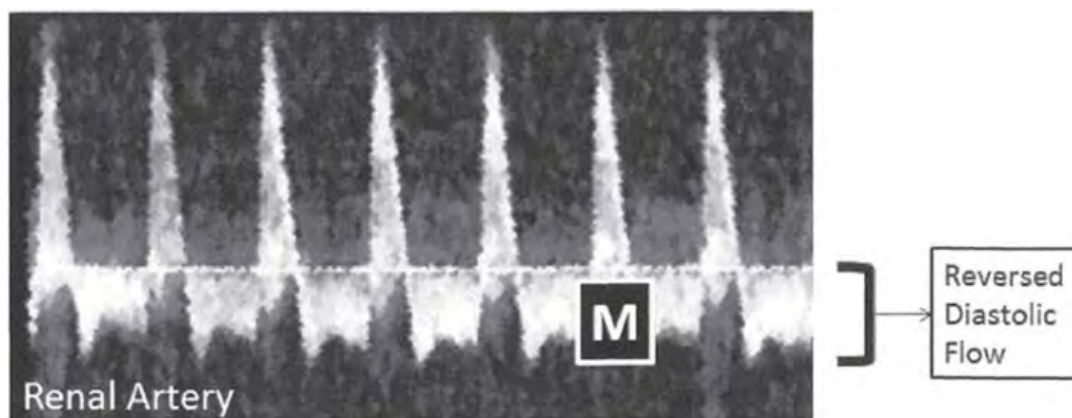
Tardus: Refers to a slowed systolic upstroke. This can be measured by acceleration time, the time from end diastole to the first systolic peak. An acceleration time >0.07 sec correlates with >50% stenosis of the renal artery

Parvus: Refers to decreased systolic velocity. This can be measured by calculating the acceleration index, the change in velocity from end diastole to the first systolic peak.

An acceleration index <3.0 m/sec- correlates with >50% stenosis of the renal artery



Renal Vein Thrombosis - This is more common in the 2nd week post transplant. Typically the kidney is swollen. Instead of showing you the Doppler of the renal vein (which would show no flow), they will most likely show you the artery, which classically has reversed diastolic flow.



Renal Vein Thrombosis- with the artery showing reversed diastolic flow. Some people call this a "reverse Msign."

Arteriovenous Fistula (AVF) - These occur secondary to biopsy. They occur about 20% of the time post biopsy, but are usually small and asymptomatic. They will likely show it with **tissue vibration artifact** (Perivascular, mosaic color assignment due to tissue vibration), with high arterial velocity, and pulsatile flow in the vein.

Pseudoaneurysm - These also occur secondary to biopsy, but are less common. They can also occur in the setting of graft infection, or anastomotic dehiscence. They will most likely show you the classic “yin-yang” color picture. Alternatively, they could show Doppler with a biphasic flow at the neck of the pseudoaneurysm.

Cancer in the Transplant

The prolonged immunosuppression therapy that renal transplant patients are on places them at significant (100x) increased risk of developing cancer.

RCC - Increased risk, with most of the cancers (90%) actually occurring in the native kidney. Etiology is not totally understood; maybe it's the immunosuppression or the fact that many transplant patients were on dialysis (a known risk factor) that leads to the cancer risk. In reality it doesn't matter, and is probably both.

Post Transplant Lymphoproliferative Disorder (PTLD) - This is an uncommon complication of organ transplant, associated with B-Cell proliferation. It is most common in the first year post transplant, and often involves multiple organs. The treatment is to back off the immunosuppression.

Cyclophosphamide - As a point of trivia, significant exposure to cyclophosphamide (less common now with the development of cyclosporin A) is associated with increased risk of urothelial cancer.

Renal Transplant Complications			
Week 1	Weeks 1-4	Months 1-6	After 6 Months
Renal Artery Thrombosis	Renal Vein Stenosis (more common)	Drug Toxicity	Chronic Rejection
Renal Artery Stenosis (more common)	Lymphocele	Lymphocele	RCC
Hematoma		Biopsy Related Injury (Pseudoaneurysm, AV Fistula)	Lymphoma
Urinoma			PTLD

Ureters

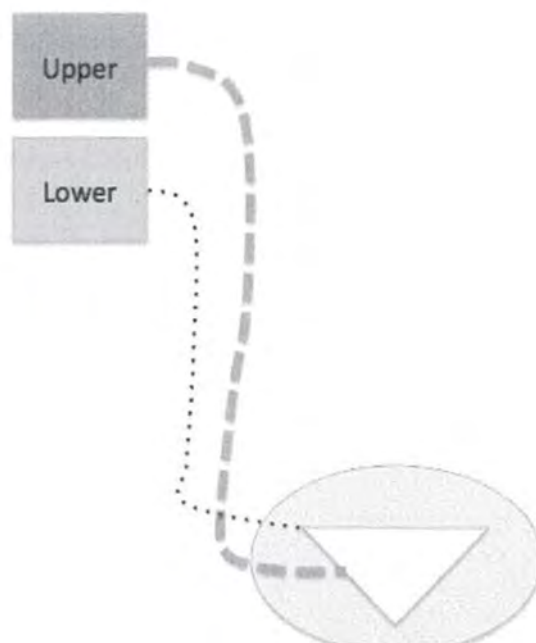
Normal Anatomy - The ureters run anterior to the psoas muscle, and empty at lateral angles of the bladder trigone. They have 3 layers, with the inner layer being transitional epithelium.

Developmental Ureter Anomalies

Congenital (primary) MEGAureter - This is a “wastebasket” term, for an enlarged ureter which is intrinsic to the ureter (as opposed to the result of a distal obstruction). Causes include (1) Distal adynamic segment (analogous to achalasia, or colonic Hirschsprungs), (2) reflux at the UVJ, (3) it just wants to be big (totally idiopathic). The distal adynamic type “obstructing primary megaureter” can have some hydro, but generally speaking an absence of dilation of the collecting system helps distinguish this from an actual obstruction. Most cases just have dilation of the lower 1/3. It is almost always unilateral (favoring the left side).

Retrocaval Ureter (circumcaval) - Although the name implies that this is the result of a maldeveloped ureter, it’s actually a developmental anomaly of the IVC. Most of the time it’s asymptomatic, but can cause partial obstruction, and recurrent UTI. IVP will show a “reverse J” or “fishhook” appearance of the ureter.

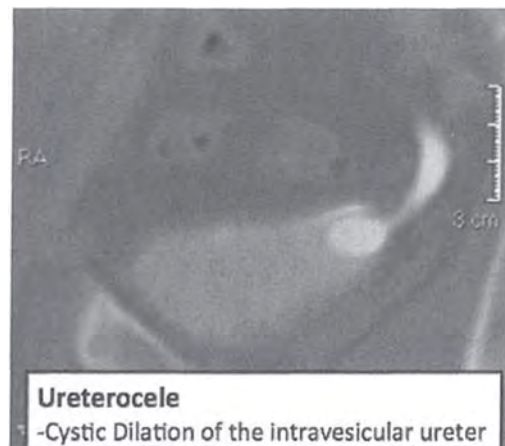
Duplicated System - The main thing to know about duplicated systems is the so called “Weigert-Meyer Rule” where the upper pole inserts inferior and medially. The upper pole is prone to ureterocele formation and obstruction. The lower pole is prone to reflux.



Weigert-Meyer Rule

- Upper Pole inserts medial and inferior

Ureterocele - A cystic dilation of the intravesicular ureter, secondary to obstruction at the ureteral orifice. IVP will show the “cobra head” sign, with contrast surrounded by a lucent rim, protruding from the contrast filled bladder. This is associated with a duplicated system (specifically the upper pole). Ureteroceles are best demonstrated during the early filling phase of the VCUG



Pseudoureterocele - This is an acquired dilation of the submucosal portion of the distal ureter. A loss of the normal lucent line around the “cobra head” suggests a pseudoureterocele. Causes include an impacted stone, a recently passed stone, and a bladder malignancy.

Ectopic Ureter - The ureter inserts distal to the external sphincter in the vestibule. More common in females and associated with incontinence (not associated with incontinence in men).

Vesicoureteral Reflux - Refers to retrograde flow of urine. The supposed malfunction is that the course of the ureter is too short as it crosses the bladder, messing with the ureteral valve mechanism. This is really a Peds thing, and discussed more in the peds chapter.

Congenital UPJ Obstruction - This is the **most common congenital anomaly of the GU tract in neonates**. About 20% of the time, these are bilateral. Most (80%) of these are thought to be caused by intrinsic defects in the circular muscle bundle of the renal pelvis. Treatment is a pyeloplasty. A Radiologist can actually add value by looking for vessels crossing UPJ prior to pyeloplasty, as this changes the management.

Things to know about UPJ Obstruction

- Most common congenital anomaly of the GU tract
- Associated with Crossing Vessels (early branching lower pole vessels compress the ureter)
- Associated with Multicystic Dysplastic Kidney on the other side.

1970 called

They want to know how to tell the difference between a prominent extrarenal pelvis vs a congenital UPJ obstruction.

The Answer is a “Whitaker Test”, which is a urodynamics study combined with antegrade pyelogram.

Ureter Infection / Inflammation

Stones - Stones tend to lodge in 3 spots: UPJ, UVJ, pelvic brim.

Ureteral Wall Calcifications - Wall calcifications should make you think about two things: (1) TB, (2) Schistosomiasis.

Ureteritis cystica - Numerous tiny subepithelial fluid filled cysts within the wall of the ureter. The condition is the result of chronic inflammation (from stones, chronic and/or infection). Typically this is seen in diabetics with recurrent UTI. There may be an increased risk of cancer.

Ureteral pseudodiverticulosis - This is similar to ureteritis cystica in that both conditions are the result of chronic inflammation (stones, infection). Instead of being cystic filling defects, these guys are multiple small outpouchings. They are bilateral 75% of the times, and favor the upper and middle third. There is an association with malignancy.

Leukoplakia - This is essentially squamous metaplasia secondary to chronic irritations (stones or infections). The bladder is more commonly involved than the ureter. Imaging findings are unlikely to be shown, but would be mural filling defects. The question is most likely this : Leukoplakia is considered premalignant and the cancer is squamous cell.

Malacoplakia - This is also an inflammatory condition that occurs in the setting of chronic UTIs (highly associated with E.Coli). It's **often seen in female immunocompromised patients**. This also has plaque-like or nodular intramural lesions, where bugs are incompletely digested. Since malacoplakia most frequently manifests as a mucosal mass involving the ureter or bladder, the most common renal finding is obstruction secondary to a lesion in the lower tract. Step 1 buzzword = Michaelis-Gutmann Bodies. Again, the bladder is more commonly affected. The question is most likely this: Malacoplakia is NOT premalignant, and usually gets better with antibiotics.

Leukoplakia = Premalignant
Malacoplakia = Not Premalignant

Retroperitoneal Fibrosis - This condition is characterized by proliferation of aberrant fibro-inflammatory tissue, which typically surrounds the aorta, IVC, iliac vessels, and frequently traps and obstructs the ureters. It is idiopathic 75% of the time. Other causes include prior radiation, medications (methyldopa, ergotamine, methysergide), inflammatory causes (pancreatitis, pyelonephritis, inflammatory aneurysm), and malignancy (desmoplastic reaction, lymphoma).

Things (trivia) you need to know:

- * *Mostly (80%) idiopathic “Ormond Disease ”*
- * *Associated with IgG4 disorders (autoimmune pancreatitis, riedels thyroid, inflammatory pseudotumor)*
- * *Classically shown with medial deviation of ureters*
- * *It’s more common in men*
- * *Malignancy associated RP fibrosis occurs about 10% of the time (some people advocate using PET to find a primary)*
- * *The Fibrosis will be Gallium avid, and PET hot in its early stages and cold in its late stages (mirroring its inflammatory stages). Metabolically active RP fibrosis will show increased FDG and Gallium uptake, regardless of a benign or malignant underlying cause*

Subcathelial Renal Pelvis Hematoma: This tends to occur in patients on long term anticoagulation. You are going to have a thickened upper tract wall - which is a classic **mimic for TCC**. They will have to show you pre and post contrast to show you that it’s **hyper dense on the pre-contrast and does NOT enhance**.

Lower Tract Cancer

Transitional Cell Carcinoma - This histologic subtype makes up a very large majority (90%) of the collecting system cancers. Imaging buzzword is “goblet” or “champagne glass sign” on CTIVP.

Risk Factors:

- O Smoking*
- o Azo Dye*
- o Cyclophosphamide*
- o Aristolochic acid (Balkan Nephropathy - see below)*
- o Horseshoe Kidney*
- o Stones*
- o Ureteral Pseudodiverticulosis*
- o Hereditary Non-Polyposis Colon Cancer (type 2)*

Some high yield statistical trivia (seriously this is high yield):

- O Ureter is the least common location for TCC of the urinary tract*
- O TCC of the renal pelvis is 2x -3x times more common than ureter*
- O TCC of the bladder is 100x times more common than ureter*
- O In the ureter 75% of the TCCs are in the bottom 1/3*
- O If you have upper tract TCC there is a 40% chance of developing a bladder TCC*
- O If you have bladder TCC there is a 4%> chance of developing a Renal Pelvis or Ureteral TCC*
- O Ureteral TCC is bilateral 5%>*



Balkan Nephropathy - This is some zebra degenerative nephropathy endemic to the Balkan States. The only reason I mention it is that it has a super high rate of renal pelvis and upper ureter TCCs. It's thought to be secondary to eating aristolochic acid (AA) in seeds of the Aristolochia clematitis plant.

Squamous cell - This is much less common than TCC (in the US anyway). The major predisposing factor is schistosomiasis (they both start with an “S”).

Hematogenous Metastasis - Mets to the ureters are rare but can occur (GI, Prostate, Renal, Breast). They typically infiltrate the periureteral soft tissues, and demonstrate Transmural involvement.

Fibroepithelial Polyp - This is a benign entity, which is usually located in the proximal ureter and produces a smooth, oblong, mobile defect on urography.

Medial Deviation “Waisting” of the Ureters	Lateral Deviation of the Ureters
Retroperitoneal Fibrosis	Retroperitoneal Adenopathy
Retrocaval Ureter (right side)	Aortic Aneurysm
Pelvic Lipomatosis	
Psoas Hypertrophy (<i>distal ureter</i>)	Psoas Hypertrophy (<i>proximal ureter</i>)

Bladder

Normal anatomy: Normal bladder is an extraperitoneal structure, with 4 layers. The dome of the bladder has a peritoneal cover. It's lined with transitional urothelium.

Developmental Anomalies

Prune Belly (Eagle Barrett) - This is a malformation triad which occurs in males. This is classically shown with a baby gram with a kid shaped like a pear (big wide belly).

Triad:

- * Deficiency of abdominal musculature
- * Hydroureteronephrosis
- * Cryptorchidism (bladder distention interferes with descent of testes)

Bladder Diverticula - These are more common in boys, and can be seen in a few situations. The “Hutch Diverticulum” occurs at the UVJ and is NOT associated with posterior urthelial valves, or neurogenic bladder. The Hutch is associated with reflux (because the normal slanted insertion of the ureter is altered). Bladder diverticulum (including the Hutch types) can be seen with Ehlers Danlos. Bladder diverticula can also be acquired secondary to chronic outlet obstruction (big prostate).

Bladder Ears - “Transitory extraperitoneal herniation of the bladder” if you want to sound smart. This is not a diverticulum. Instead, it's transient lateral protrusion of the bladder into the inguinal canal. It's very common to see, and likely doesn't mean crap. However, some sources say an inguinal hernia may be present 20% of the time. Smooth walls, and usually wide necks can help distinguish them from diverticula.

Cloacal Malformation - GU and GI both drain into a common opening (like a bird). This only happens in females.

Urachus - The umbilical attachment of the bladder (initially the allantois then urachus) usually atrophies and becomes the umbilical ligament (as the bladder descends into the pelvis). A persistent patent urachus can result in urine flow from the bladder to the umbilicus (and then likely someone's unsuspecting face). There is a spectrum of these things from patent -> sinus -> diverticulum -> Cyst. They can get infected. Really **the main thing to know is that they get adenocarcinoma**. It's midline and they get adenocarcinoma.

Bladder Cancer

Rhabdomyosarcoma - This is the most common bladder cancer in humans less than 10 years of age. They are often infiltrative, and it's hard to tell where they originate. "Paratesticular Mass" is often a buzzword. They can met to the lungs, bones, and nodes. The Botryoid variant produces a polypoid mass, which looks like a bunch of grapes.

Bladder Cancer

Typical = Transitional Cell
Schistosomiasis = Squamous
Urachus = Adenocarcinoma
Kids = Rhabdomyosarcoma

Transitional Cell Carcinoma - As stated above, the bladder is the most common site, and this is by far the most common subtype. All the risk factors, are the same as above. If anyone would ask "superficial papillary" is the most common TCC bladder subtype.

Squamous Cell Carcinoma - When **1** say Squamous Cell Bladder, you say Schistosomiasis. This is convenient because they both start with an "S." The classic picture is a heavily calcified bladder and distal ureters (usually shown on plain film, but could also be on CT).

Adenocarcinoma of the Bladder - This is a common trick question. When **I** say Adenocarcinoma of the Bladder, you say Urachus. 90% of urachal cancers are located midline at the bladder dome.

Leiomyoma - Benign tumor (not cancer). It's often incidentally discovered (most common at the trigone). If anyone asks it's the most common mesenchymal bladder tumor.

Diversion Surgery

After radical cystectomy for bladder cancer there are several urinary diversion procedures that can be done. People generally group these into incontinent and continent procedures. There are a ton of these (over 50 have been described). I just want to touch on the big points, and focus on complications (the most testable subject matter). The general idea is that a piece of bowel is made into either a conduit or reservoir, and then the ureters are attached to it.

Early Complications:

- Alteration in bowel function:* **Adynamic ileus is the most common early complication**, occurring in almost 25% of cases. In about 3% of cases you can get SBO, usually from adhesive disease near the enteroenteric anastomosis.
- Urinary Leakage:* This occurs in about 5% of cases, and usually at the ureteral-reservoir anastomosis. A urinoma can develop when the leaked urine is not collected by urinary drains.
- Fistula:* This is uncommon and seen more in patients who have had pelvic radiation.

Late Complications (> 30 days)

- Urinary infection:* This can be early or late.
- Stones:* Remember to look on the non-contrasted study.
- Parastomal Herniation:* This occurs about 15% of the time with ileal conduits. Obesity is a contributing factor. Most don't matter, but 10% will need a surgical fix.
- Urinary stricture:* The **left side is higher risk than the right**, secondary to the angulation (it's brought through or under the mesentery).
- Tumor Recurrence:* The more advanced the original disease, the higher the risk for recurrence. The incidence is between 3-15%, and can present as a soft tissue mass at the ureter, bladder, or a pelvic lymph node.

Infectious / Inflammation

Emphysematous Cystitis - Gas forming organism in the wall of the bladder. More than half the time it's a diabetic patient. It's usually from E. Coli. It's gonna be very obvious on plain film and CT. Ultrasound would be sneaky, and you'd see dirty shadowing.

TB - The upper GU tract is more commonly effected, with secondary involvement of the bladder. Can eventually lead to a thick contracted bladder. Calcifications might be present.

Schistosomiasis - Common in the third world. Eggs are deposited in the bladder wall which leads to chronic inflammation. Things to know: the entire bladder will calcify (often shown on plain film or CT), and you get squamous cell cancer.

Fistula - This occurs basically in 3 conditions; (1) diverticulitis, (2) Crohns, (3) Cancer. They are more common in men, although women are at significantly increased risk after hysterectomy (the uterus protects the bladder).

Most Common Cause

- Colovesical Fistula = Diverticular Disease
- Ileovesical Fistulas = Crohns
- Rectovesical Fistulas = Neoplasm or Trauma

Neurogenic Bladder - This comes in two flavors: (a) small contracted bladder, (b) atonic large bladder. The buzzword / classic sign is "*pine cone*" bladder, because of its appearance. It can lead to urine stasis, and that stasis can predispose to bladder CA, **stones**, and infection.

Acquired Bladder Diverticula - As mentioned above, these can be acquired mainly via outlet obstruction (just think big prostate). They are most common at the UVJ. They can lead to stasis, and that stasis can predispose to bladder CA, stones, and infection.

Bladder Stones - These guys show up in two scenarios: (1) they are bom as kidney stones and drop into the bladder (2) they develop in the bladder secondary to stasis (outlet obstruction, or **neurogenic bladder**). They can cause chronic irritation and are a known risk factor for both TCC and SCC.

"Pear Shaped Bladder" - This is more of a sign than a pathology. Think two things (1) pelvic lipomatosis, and (2) hematoma.

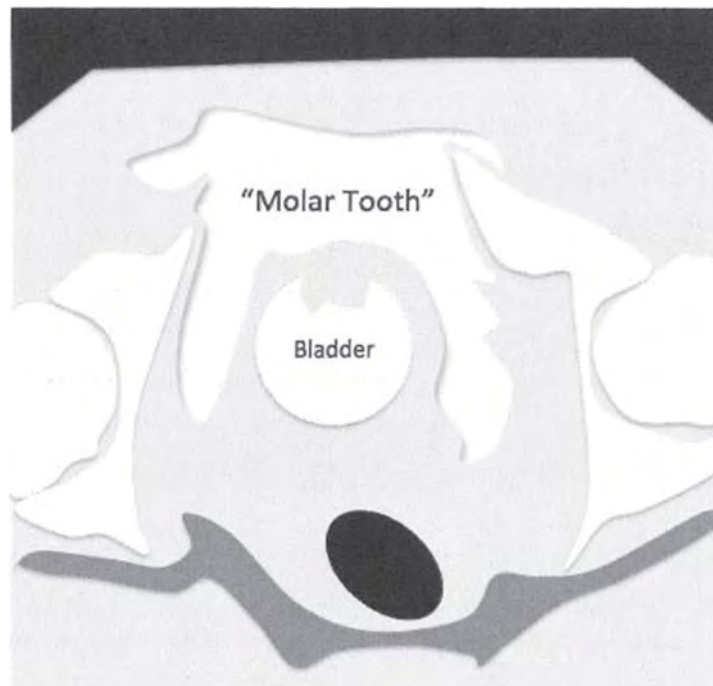
Bladder Trauma

Bladder Rupture - What they want you to know is; extra versus intra peritoneal rupture. CT Cystography (contrast distending bladder) is needed.

* *Extraperitoneal*-This one is **more common**. Almost always associated with pelvic fracture. This can be managed medically.

- o If there is a pelvic fracture, then the chance of a bladder rupture is 10%.
- o If there is a bladder rupture, there is almost always a pelvic fracture

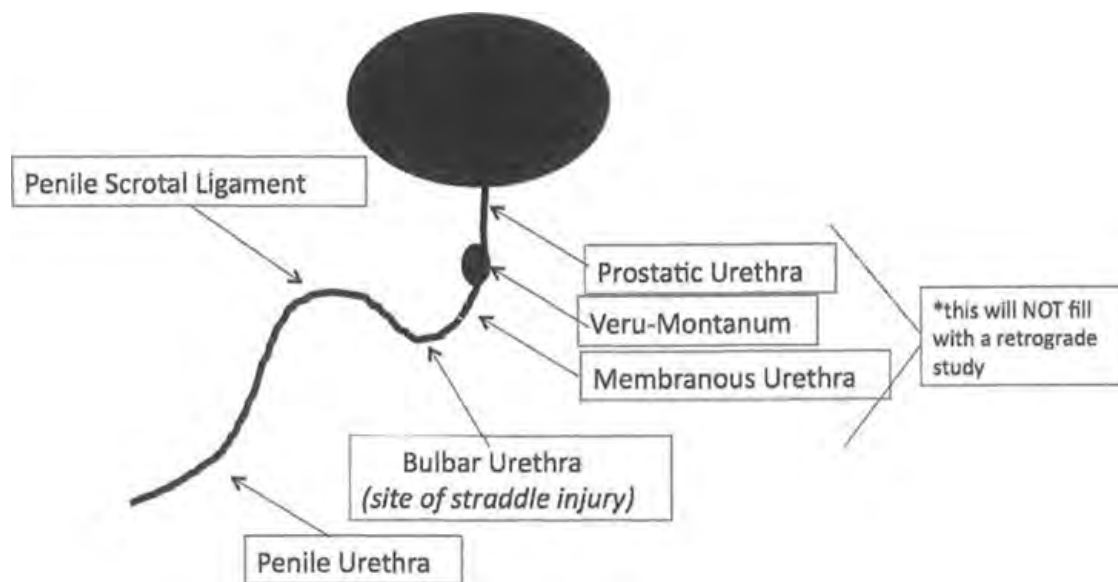
o Molar Tooth Sign: Contrast surrounding the bladder, in the prevesicle space of Rezius. This indicated extraperitoneal bladder rupture. •



- *Intraperitoneal* This one is less common. Direct blow to a full bladder, basically pops the balloon and blows the top off (bladder dome is the weakest part). The dude will have contrast outlining bowel loops and in the paracolic gutters. This requires surgery.

The Urethra

Male:



Normal Anatomy (most commonly seen on a RUG), is high yield.

There are really only about three things to know other than anatomy: Urethral Injury, Urethrorectal fistula, Infection, and Diverticulum (with cancer implications).

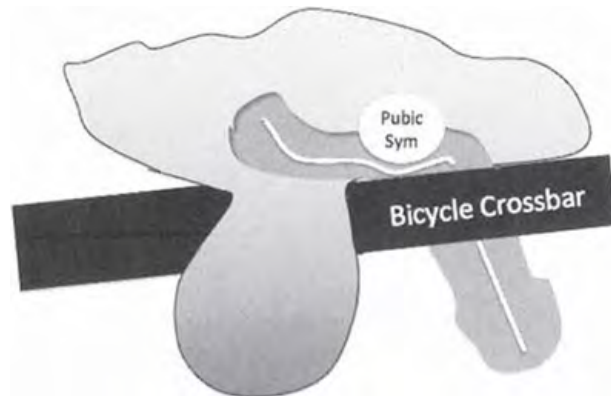
Trauma: Urethral injuries are graded based on their location (1-5).

- * *Type 1: Stretched - periurethral hematoma would be present. This is not well seen on RUG.*
- * *Type 2: Rupture ABOVE the UG diaphragm. Extraperitoneal contrast is present.*
- * *Type 3: Rupture BELOW the UG diaphragm. Extraperitoneal and perineal contrast is present.*
- * *Type 4: Injury involves the bladder extending to the urethra.*
- * *Type 5: There is injury to the anterior urethra.*

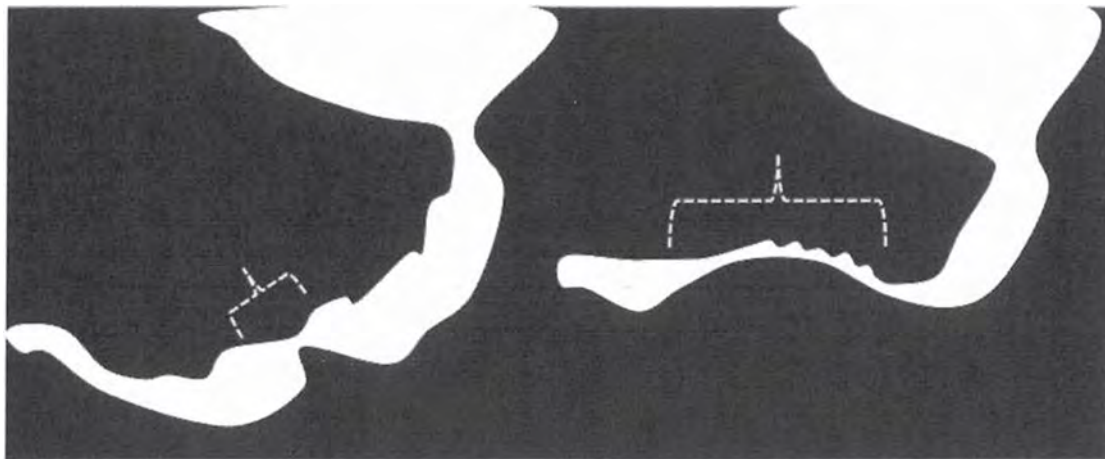
Urethral Strictures -

The Bicycle Crossbar Injury *vs* The Injury From a Woman of Questionable Moral Standard

Straddle Injury: The most common external cause of traumatic stricture is this type of mechanism. The physiology is compression of the urethra against the inferior edge of the pubic symphysis. **The bulbous urethra is the site of injury** (this is the most likely question).



Gonococcal Urethral Stricture: This tends to be a **long irregular stricture** (the straddle stricture was short). It occurs in the **distal bulbous urethra**.



Straddle Injury Gonococcal
Short Segment - Bulbous Urethra Long Segment & Irregular - Bulbous Urethra

Other Male Urethral Pathologies:

Pancreatic Transplant: This has been known to cause urethral injury, if the drainage is to the bladder (the old way of doing it). Extravasation from urethral injury is said to occur in about 5% of cases, and is secondary to pancreatic enzymes jacking the urethra.

Condyloma Acuminata - Multiple small filling defects seen on a RUG should make you think this. Although, instrumentation including a retrograde urethrography is actually not recommended because of the possibility of retrograde seeding.

Urethrectal Fistula: This may occur post radiation, and is classically described with brachytherapy (occurs in 1% of patients).

Urethral Diverticulum: In a man, this is almost always the result of long term foley placement.

Cancer: Malignant tumors of the male urethra are rare. When they do occur **80% are squamous cell cancers** (the exception is that prostatic urethra actually has transitional cell 90% of the time).

Urethral Diverticulum Cancer: Cancer in a urethral diverticulum is nearly **ALWAYS adenocarcinoma** (rather than squamous cell).

Female:

Female Urethral Diverticulum: Urethral diverticulum is way more common in females. They are usually the result of repeated infection of the periurethral glands (classic **history is “repeated urinary tract infections”**). In case books and conferences this is **classically shown as a Sagittal MRI**. It often coexists with stress urinary incontinence (60%) and urinary infection. The **buzzword is “saddle-bag”** configuration, which supposedly is how you tell it from the urethra. Stones can also develop in these things. All this infection and irritation leads to **risk of cancer**, and the very common **high yield factoid is this is most commonly adenocarcinoma (60%)**.

GU Cancer Blitz!

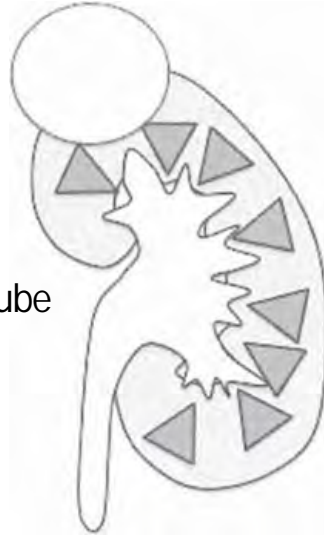
Renal Cancer: *(Adenocarcinoma)*

— Subtypes

- Clear Cell - most common (*enhances more*)
- Papillary - 2nd most common (*enhances less*)
- Medullary - Buzzword for Sick Cell Trait
- Chromophobe - Buzzword for Burt Hogg Dube

— Syndrome

- Von Hippie Lindau - Multiple Clear Cells



Ureter Cancer *(Transitional Cell)*:

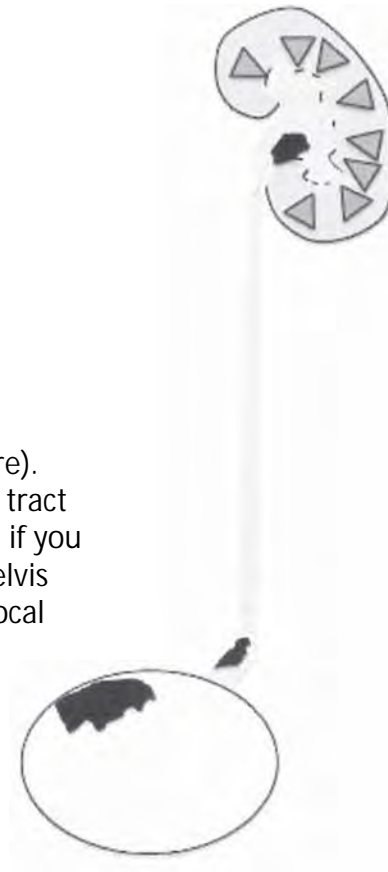
— Location — *think about where you get the most stasis*

- Renal Pelvis- Twice as common as Ureter
- Distal Third of the Ureter - Most common site
- Middle is 2nd, and Proximal is 3rd

— Relationship to Bladder CA:

Bladder CA is way more common (like 100x more).
So if you have bladder CA you don't need upper tract
CA. Since upper tract CA is not all that common, if you
smoked enough Marlboro Reds to get a renal pelvis
CA, you probably smoked enough to get multi focal
disease including the bladder.

Bladder can be isolated
Ureteral usually has bladder



Bladder Cancer



Transitional Cell CA

- The normal kind
- Much more common than the ureter (like 100x)

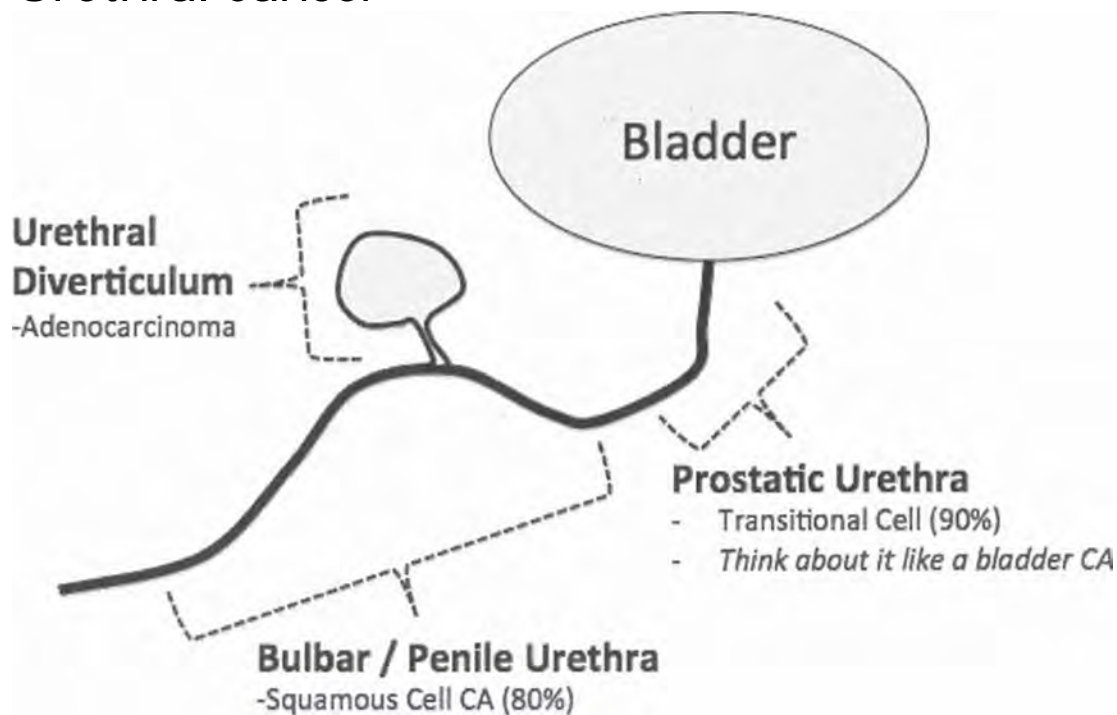
Squamous Cell CA

- With lots of calcifications,
- In the setting of schistosomiasis

Adenocarcinoma

- Midline CA
- Patent Urachus

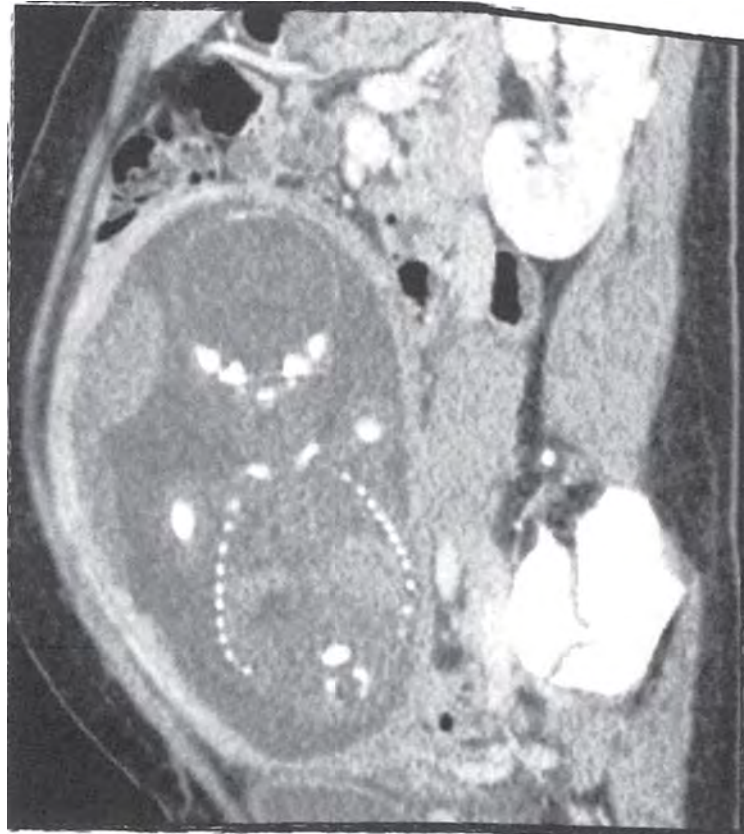
Urethral Cancer



4

Reproductive

Prometheus Lionhart, M.D.



This section can essentially only be tested in ultrasound and MRI (mostly ultrasound). It has the potential to be image rich (even more so than other sections). I would know the sonographic features for everything I mention in this chapter, and to a lesser extent, the MR imaging features.

High Yield Topics:

- Anatomy is always high yield
- Differentiating Ovarian Masses
- Infertility work up (males and females)
- Fetal MRI (posterior valves, double bubble, diaphragm hernia).

Ladies First - *Uterus and Vagina*

The Uterus - Changes During Life

In the neonate the uterus is supposed to be prominent, as mom's hormones and placental hormones are still having some influence. The cervix will be bigger than the fundus. The prepubertal uterus takes on a more tubular shape, and the cervix is about the same size as the fundus. In puberty, uterus begins to have the adult "pear shaped" configuration, and the fundus will be larger than the cervix. During puberty (but not really before it) you will see an endometrium, and it will vary in phases during the menstrual cycle.

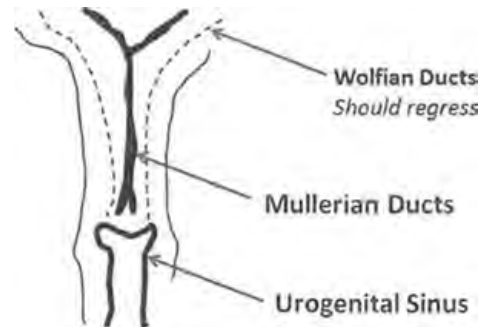
The Ovaries - Changes During Life

Just like with the uterus, infants tend to have larger ovaries (volume around 1cc), which then decreases and remains around or less than 1cc until about age 6. The ovaries then gradually increase to normal adult size as puberty approaches and occurs.

Turner Syndrome - The XO kids. Besides often having aortic co-arcuations, and horseshoe kidneys they will have a pre-puberty uterus and streaky ovaries.

Embryology

The quick and dirty of it is that the mullerian ducts make the ovaries, and upper 2/3 of the vagina. The urogenital sinus grows up to meet the mullerian ducts and makes the bottom 1/3 of the vagina. Wolfian ducts are the boy parts, and should regress completely in girls.



Vocab

*(in case you don't speak French or
whatever)*

Cornus = Uterus

Collis = Cervical

Mullerian Anomalies:

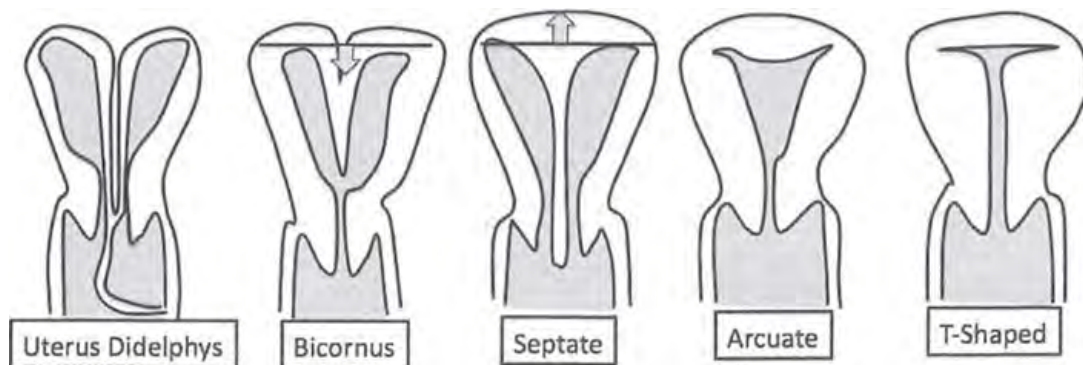
As described above, the upper two thirds of the vagina is formed by the mullerian ducts, and the lower one third is formed from the urogenital sinus. Mullerian anomalies are broken into three groups: Disorders of Dysgenesis, Lateral Fusion, and Vertical Fusion.

Disorders of Dysgenesis:

- * **Mullerian Agenesis** (Mayer-Rokitansky-Kuster-Hauser syndrome): Has three features: (1) vaginal atresia , (2) absent or rudimentary uterus (unicornuate or bicornuate) and (3) normal ovaries. The key piece of trivia is that the **kidneys have issues** (agenesis, ectopia) in about half the cases.
- * **Unicornuate Uterus** - There are 4 subtypes (basically +/- rudimentary horn, +/- endometrial tissue). Obviously, endometrial tissue in a non-communicating horn is going to cause pelvic pain. Also, Endometrial tissue in a rudimentary horn (communicating or not - increases the risk of miscarriage and uterine rupture). **40% of these chicks will have renal issues (usually renal agenesis) ipsilateral to the rudimentary horn.**

Disorders of Lateral Fusion (duplication defects):

Lateral fusion defects result in duplications or partial duplications, from impaired fusion and or septal resorption.



Uterus Didelphys - This is a complete uterine duplication (two cervices). If the patient does not have vaginal obstruction this is usually asymptomatic.

Bicornus - This comes in two flavors (one cervix unicollis, two cervix bicollis). There will be separation of the uterus by a deep myometrial cleft. Although they can have an increased risk of fetal loss, it's much less of an issue compared to Septate.

Septate - This one has two endometrial canals separated by a fibrous (or muscular) septum. Fibrous vs Muscular can be determined with MRI and this distinction changes surgical management (different approaches). There is an increased risk of infertility and recurrent spontaneous abortion. They can resect the septum.

Bicornuate vs Septate - this requires some kind of cross sectional imaging (you can't tell by HSG). You distinguish the two by the apex of the fundal contour:

- * Apex of Fundal Contour > 5 mm Above Tubal Ostia = Septate*
- * Apex of Fundal Contour < 5mm Above Tubal Ostia = Bicornuate*
- * Other important trivia is; Septate has established increased 1st trimester loss, bicornuates have alot fewer problems.*

Arcuate Uterus - Mild smooth concavity of the uterine fundus (instead of normal straight or convex) This is not really a malformation, but more of a normal variant. It is **NOT associated with infertility or obstetric complications.**

T- Shaped - This is the **DES related anomaly**. It is historical trivia, and therefore extremely high yield for the "exam of the future." DES was a synthetic estrogen given to prevent miscarriage in the 1940s. The daughters of patients who took this drug ended up with vaginal clear cell carcinoma, and uterine anomalies - classically the "T-Shaped Uterus."

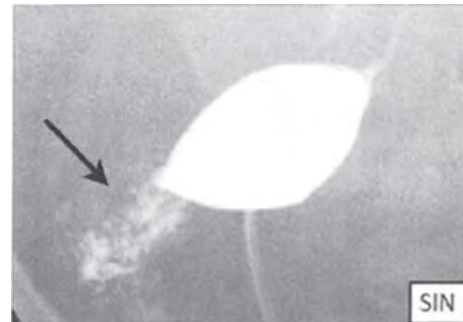
Disorders of Vertical Fusion (canalization defects):

Vertical fusion anomalies occur when the upper 2/5 fails to fuse normally with the bottom 1/3 (sinovaginal bulb).

- * **Cervical Dysgenesis:** This can happen.*
- * **Transverse Vaginal Septa:** This thing is most common in the upper vagina, and that is probably more than you need to know about it.*

Acquired Pathology

Salpingitis Isthmica Nodosa (SIN): This is a nodular scarring of the fallopian tubes that produces an Aunt Minnie Appearance. As trivia, it usually involves the proximal 2/3 of the tube. This is of unknown etiology, but likely post inflammatory / infectious. It's strongly **associated with infertility and ectopic pregnancy** and that is likely the question.



Uterine AVM - These can be congenital or acquired, with acquired types being way more common. They can be serious business and you can totally bleed to death from them. The typical ways to acquire them include: **previous dilation and curettage**, therapeutic abortion, caesarean section, or just multiple pregnancies. Doppler ultrasound is going to show: serpiginous and/or tubular anechoic structures within the myometrium with **high velocity color Doppler flow**.

Intrauterine Adhesions (Ashermans) - This is scarring in the uterus, that occurs secondary to injury: prior dilation and curettage, surgery, pregnancy, or infection (classic GU TB). This is typically shown on HSG, with either (a) non filling of the uterus, or (b) multiple irregular linear filling defects (lacunar pattern), with inability to appropriately distend the endometrial. MRI would show a bunch of T2 dark bands. Clinically, this results in infertility.



Endometritis - This is in the spectrum of PID. You often see it 2-5 days after delivery, especially in women with prolonged labor or premature rupture. You are going to have fluid and a thickened endometrial cavity. You can have gas in the cavity (not specific in a post partum women). It can progress to pyometrium, which is when you have expansion with pus.

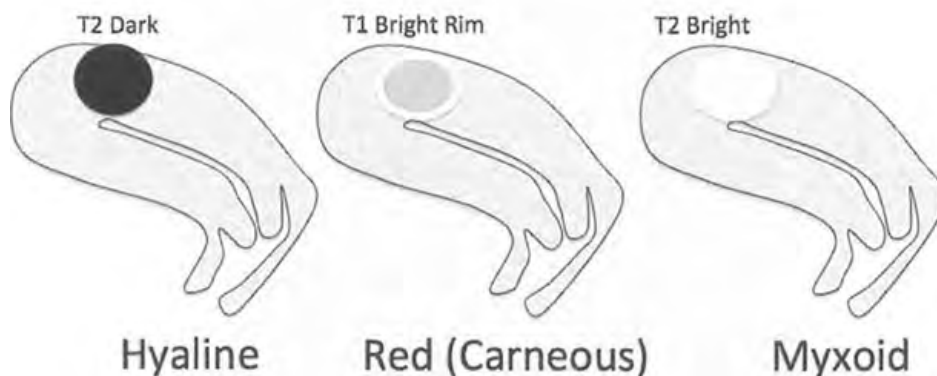
Masses and Tumors and Stuff

Fibroids (Uterine Leiomyoma): These benign smooth muscle tumors are the most common uterine mass. They are more common in women of African ancestry. They like estrogen and are most common in reproductive age (rare in prepubertal females). Because of this estrogen relationship they tend to grow rapidly during pregnancy, and involute with menopause. Their location is classically described as submucosal (least common), intramural (most common), or subserosal.

Typical Appearance: The general rule, is they **can look like anything**. Having said that, they are usually hypoechoic on ultrasound, often with peripheral blood flow and shadowing in the so called “Venetian Blind” pattern. On CT, they often have peripheral calcifications (“popcorn” as seen on plain film). On MRI, **T1 dark** (to intermediate), **T2 dark**, and variable enhancement. The fibroids with higher T2 signal are said to respond better to IR treatment. A variant subtype is the lipoleiomyoma, which is fat containing.

Degeneration: 4 types of degeneration are generally described. *What they have in common is a lack of / paucity of enhancement (fibroids normally enhance avidly).*

- O *Hyaline* - This is the most common type. The fibroid outgrows its blood supply, and you end up getting the accumulation of proteinaceous tissue. They are T2 dark, and do not enhance post Gd.
- O *Red (Carneous)* - This one **occurs during pregnancy**. This is the cause of venous thrombosis. The classic imaging finding is a **peripheral rim of T1 high signal**. The T2 signal is variable.
- O *Myxoid*- Uncommon type of degeneration. This is suggested by T1 dark, **T2 bright** and minimal gradual enhancement.
- o *Cystic* - Uncommon type -

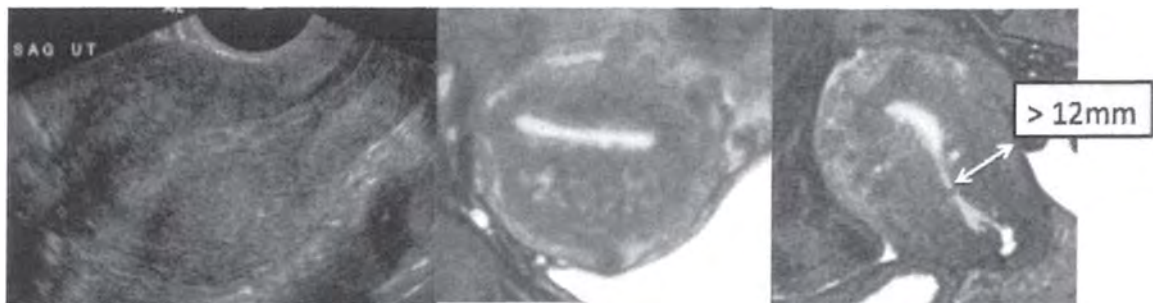


Uterine Leiomyosarcoma - The risk of malignant transformation to a leiomyosarcoma is super low (0.1%). These look like a fibroid, but rapidly enlarging. Areas of necrosis are often seen.

Adenomyosis - This is endometrial tissue that has migrated into the myometrium. You see it **most commonly in multiparous women of reproductive age, especially if they've had a history of uterine procedures** (Caesarian section, dilatation and curettage).

Although there are several types, adenomyosis is usually generalized, favoring large portions of the uterus (especially the **posterior wall**), but **sparing the cervix**. It classically causes marked enlargement of the uterus, with preservation of the overall contour.

They can show it with Ultrasound or MRI. Ultrasound is less specific with findings including a heterogeneous uterus (hyperechoic adenomyosis, with hypoechoic muscular hypertrophy), or just enlargement of the posterior wall. MRI is the way better test with the most classic feature being **thickening of the junctional zone of the uterus to more than 12 mm** (normal is < 5mm). The thickening can be either focal or diffuse. Additionally, the findings of small high T2 signal regions corresponding to regions of cystic change is a classic finding.



Adenomyosis of the Uterus
- Note the T2 bright cystic foci, and thick junctional zone

Thick Endometrium

Remember the stripe is measured without including any fluid in the canal. Focal or generalized thickening in post menopausal women greater than 5mm should get sampled. Premenopausal endometriums can give very thick - up to 20mm can be normal.

Trivia:

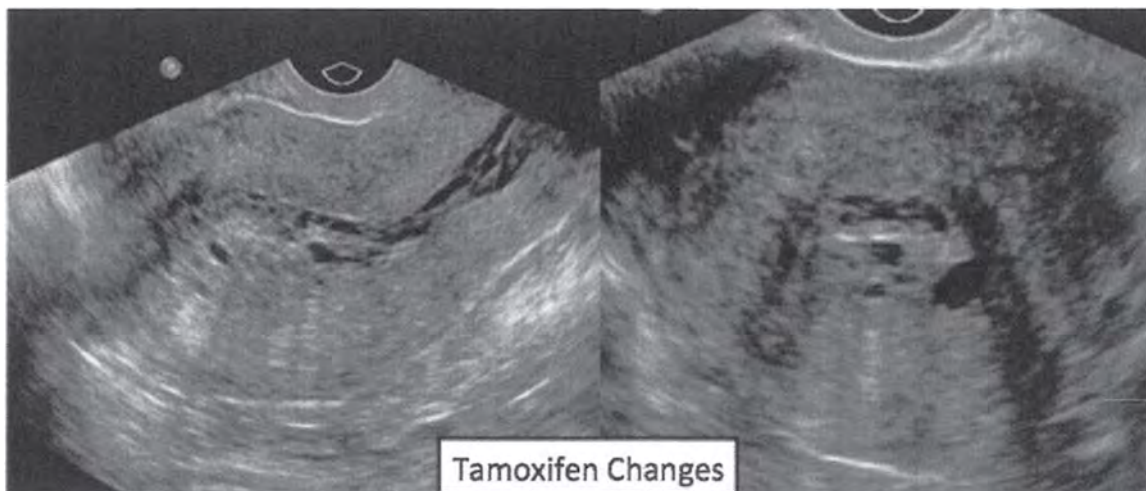
- * *Estrogen secreting tumors - Granulosa Cell tumors of the ovary will thicken the endometrium.*
- * *Hereditary Non-Polyposis Colon Cancer (HNPCC) - have a 30-50% increased risk of endometrial cancer*

Post Menopausal Bleeding:

Is it from atrophy or cancer?

- Endometrium less than 5mm = Probably Atrophy
- Endometrium > 5mm = Maybe cancer and gets a biopsy

Tamoxifen Changes - This is a SERM (acts like estrogen in the pelvis, blocks the estrogen effects on the breast). It's used for breast cancer, but **increases the risk of endometrial cancer**. It will cause subendometrial cysts, **and the development of endometrial polyps** (30%). Normally post menopausal endometrial tissue shouldn't be thicker than 4mm, but on Tamoxifen the endometrium gets a pass up to 8mm. At >8mm it gets a biopsy. If you are wondering if a polyp is hiding you can get a sonohysterogram (ultrasound after instillation of saline).



Endometrial Fluid - In a premenopausal women this is a common finding. In a post menopausal women it means either cervical stenosis or an obstructing mass (**usually cervical stenosis**).

Cervix

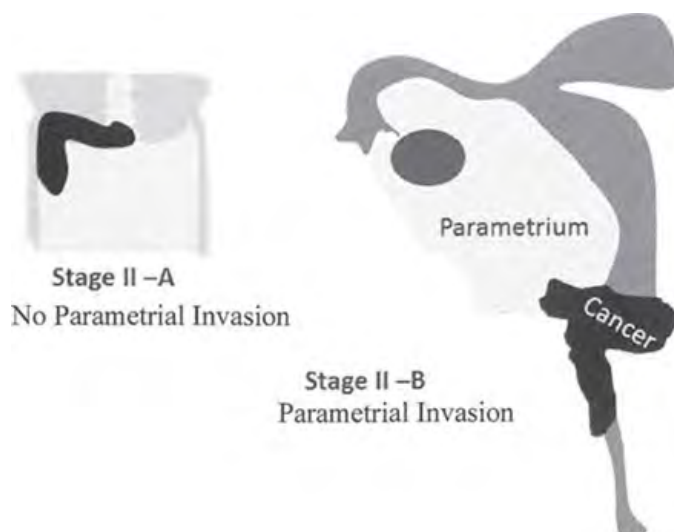
Cancer: It's **usually squamous cell**, related to **HPV** (like 90%). The big thing to know is parametrial invasion (stage lib). Stage IIa or below is treated with surgery. **Once you have parametria! invasion (stage lib)**, or involvement of the lower **1/3** of the vagina it's gonna get chemo/ radiation. In other words, management changes so that is the most likely test question.

Cervical Cancer Staging Pearls		
Stage II A	Spread beyond the cervix, but NO parametrial invasion	Surgery
Stage II B	Parametrial involvement but does NOT extend to the pelvic side wall.	Chemo/ Radiation
Stage III A	Involves the lower third of the vagina but NOT the pelvic sidewall and does NOT obstruct the ureters or invade adjacent organs.	Chemo/ Radiation
Stage III B	Involves the pelvic sidewall or causes hydronephrosis	Chemo/ Radiation

What is this Parametrium ?

The parametrium is a fibrous band that separates the supravaginal cervix from the bladder. It extends between the layers of the broad ligament.

The uterine artery runs inside the parametrium, hence the need for chemo - once invaded.



Nabothian Cysts - These are usually on the cervix and you see them all the times. They are the result of inflammation causing epithelium plugging of mucous glands.

Vagina

Solid Vaginal Masses:

A solid vaginal mass is usually a bad thing. It can be secondary (cervical or uterine carcinoma protruding into the vagina), or primary such as a clear cell adenocarcinoma or rhabdomyosarcoma.

Leiomyoma - Rare in the vagina, but can occur (most commonly in the anterior wall).

Squamous Cell Carcinoma - The most common cancer of the vagina (85%). This is associated with HPV. This is just like the cervix.

Clear Cell Adenocarcinoma - This is the zebra cancer seen in women whose mothers took DES (a synthetic estrogen thought to prevent miscarriage). That plus “T-Shaped Uterus” is probably all you need to know.

Vaginal Rhabdomyosarcoma - This is the most common tumor of the vagina in children. There is a bimodal age distribution in ages (2-6, and 14-18). They usually come off the anterior wall near the cervix. It can occur in the uterus, but typically invades it secondarily. Think about this when you see a solid T2 bright enhancing mass in the vagina / lower uterus in a child.

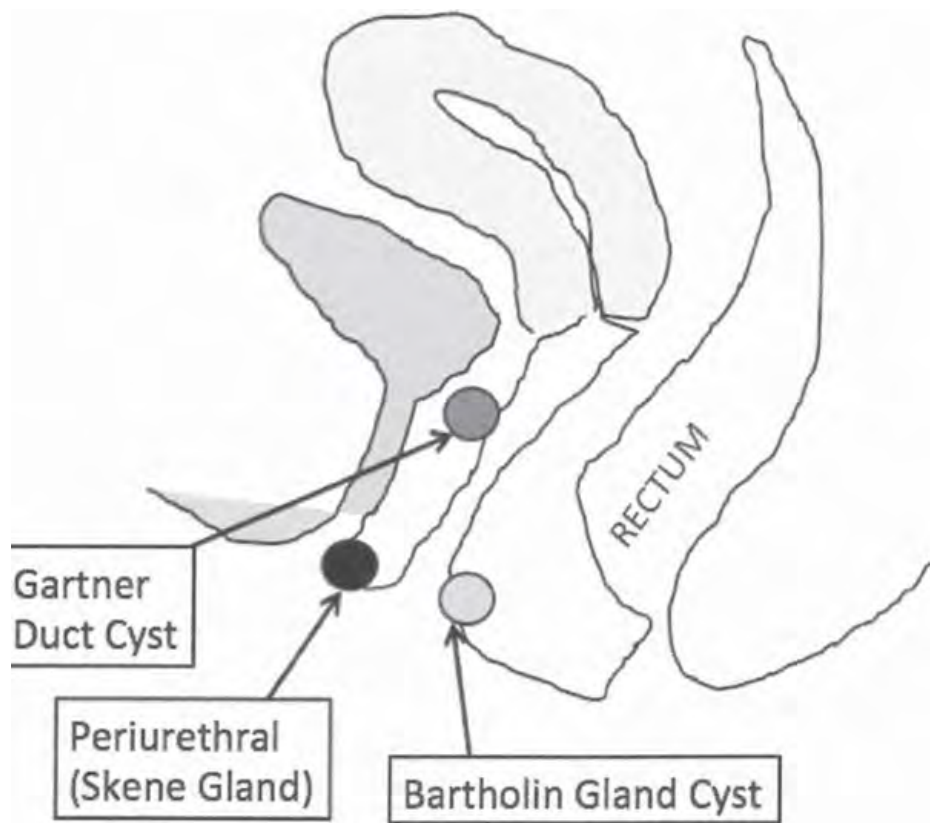
Mets Trivia: A met to the vagina in the anterior wall upper 1/3 is “always” (90%) upper genital tract. A met to the vagina in the posterior wall lower 1/3 is “always (90%) from the GI tract.

Cystic Vaginal Masses:

Gartner Ducts Cysts - These are the result of **incomplete regression of the wolffian ducts**. They are classically located along the anterior lateral wall of the upper vagina. If they are located at the level of the urethra, that **can cause mass effect on the urethra** (and symptoms).

Bartholin Cysts - These are the result of obstruction of the Bartholin glands (mucin-secreting glands from the urogenital sinus). They are found below the pubic symphysis (helps distinguish them from Gartner duct).

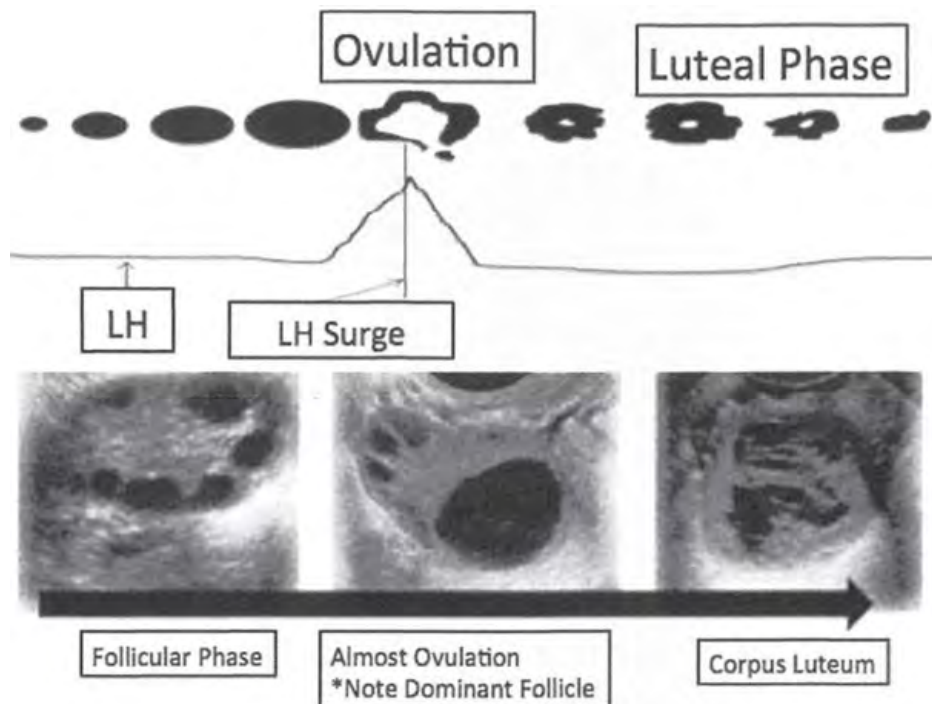
Skene Gland Cysts - Cysts in these periurethral glands, can cause recurrent UTIs and urethral obstruction.



Ovaries /Adnexa

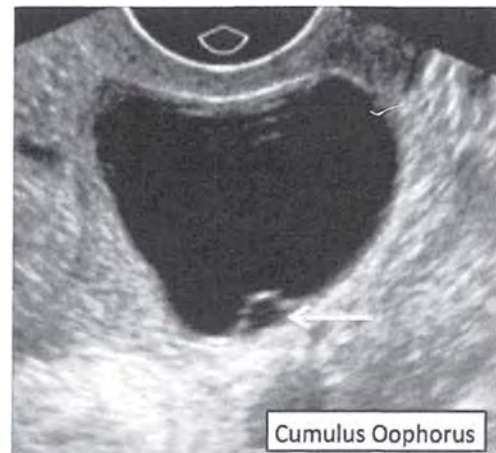
- Before I start, a few general tips (1) never biopsy or recommend biopsy of an ovary, (2) on CT if you can't find the ovary, follow the gonadal vein, and (3) hemorrhage in a cystic mass usually means it's benign.
- A quick note on ovarian size, the maximum ovarian volume can be considered normal up until 15ml (some say 20ml). The post menopausal ovary should NOT be larger than 6cc.

Let's talk about ovulation, *to help understand the normal variation in the ovary.*



Follicles seen during the early menstrual cycle are typically small (< 5mm in diameter). By day 10 of the cycle, there is usually one follicle who has emerged as the dominant follicle. By mid cycle, this dominant follicle has gotten pretty big (around 20mm). Its size isn't surprising because it contains a mature ovum. The LH surge causes the dominant follicle to rupture releasing the egg. The follicle then regresses in size forming a Corpus Luteum. A small amount of fluid can be seen in the cul-de-sac. Occasionally, a follicle bleeds and re-expands (hemorrhagic cyst) - more on this later.

Cumulus Oophorus - this is a piece of anatomy trivia. It is a collection of cells in a mature dominant follicle that protrudes into the follicular cavity, and **signals imminent ovulation** (its absence means nothing).



Let's speak briefly about fertility meds

Medications such as a Clomiphene Citrate (Clomid), force the maturation of multiple bilateral ovarian cysts. It is not uncommon for the ovaries of women taking this drug to have multiple follicles measuring more than 20mm in diameter by mid cycle.

Theca Lutein Cysts - this is a type of functional cyst (more on that below), related to overstimulation from b-HCG. What you see are large cysts (~ 2-3 cm) and the **ovary has a typical multilocular cystic “spoke-wheel” appearance.**

Think about 3 things:

- Multifetal pregnancy;*
- Gestational trophoblastic disease (moles), and*
- Ovarian Hyperstimulation syndrome.*



Ovarian Hyperstimulation Syndrome - This is a complication associated with fertility therapy (occurs in like 5%). They will show you the ovaries with **theca lutein cysts**, then ascites, and **pleural effusions**. They may also have pericardial effusions. Complications include increased risk for ovarian torsion (big ovaries), and hypovolemic shock.

Old vs Young

Premenstrual: The ovaries of a pediatric patient stay small until around age 8-9. Ovaries may contain small follicles.

Premenopausal: A piece of trivia; premenopausal ovaries **may be HOT on PET** (depending on the menstrual cycle). This is why you do a PET in the first week of the menstrual cycle.

Postmenopausal (*oneyear after menses stops*): Considered **abnormal if** it exceeds the upper limit of normal, or is **twice the size of the other ovary (even if no mass is present)**. Small cysts (< 3cm) are seen in around 20% of post menopausal women. In general, postmenopausal ovaries are atrophic, lack follicles, and can be difficult to find with ultrasound. The ovarian volume will decrease from around 8cc at age 40, to around 1cc at age 70. The **maximum ovarian volume in a post menopausal woman is 6ml**. Unlike premenopausal ovaries, **post menopausal ovaries should NOT be hot on PET**.

Cyst in Postmenopausal Woman *WTF Do I Do Now?*

If the cyst is simple, regardless of age it's almost certainly benign.

Having said that, the rule is:

- Greater than 1cm gets yearly follow up
- Greater than 7cm gets either an MRI or a surgeon.

If it's first seen on another modality (CT), I would get an Ultrasound first to confirm its totally cystic - without suspicious features like papillary projections, nodules, thick septations etc.... Then follow the rules as above.

Smokey this is not 'Nam, this is bowling. There are rules...

The Big 6

(The Sinister Six - For You Spiderman Fans)

In most clinical practices, the overwhelming majority of ovarian masses are benign (don't worry I'll talk about cancer too).

- Physiologic and functioning follicles
- Corpora lutea
- Hemorrhagic cysts
- Endometriomas
- Benign cystic teratomas (dermoids)
- Polycystic ovaries

Functioning Ovarian Cysts: Functioning cysts (follicles) are affected by the menstrual cycle (as I detailed eloquently above). These cysts are benign and usually 25mm or less in diameter. They will usually change / disappear in 6 weeks. If a cyst persists and either does not change or increases in size, it is considered a nonfunctioning cyst (not under hormonal control).

Simple cysts that are > 7cm in size may need further evaluation with MR (or surgical evaluation). Just because it's hard to evaluate them completely on US when they are that big, and you risk torsion with a cyst that size.

Corpus Luteum: The normal corpus luteum arises from a dominant follicle (as I detailed eloquently above). These things can be large (up to 5-6cm) with a variable appearance (solid hypoechoic, anechoic, thin walled, thick walled, cyst with debris). The most common appearance is solid and hypoechoic with a "ring of fire" (intense peripheral blood flow).

Corpus Luteum vs Ectopic Pregnancy

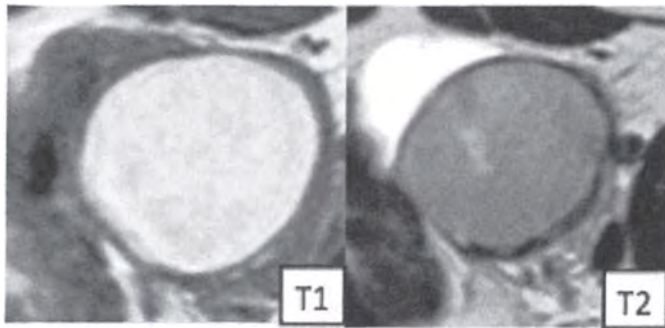
They both can have that “ring of fire” appearance, but please don’t be an idiot about this. Most ectopic pregnancies occur in the tube (the corpus luteum is an ovarian structure). If you are really confused, a differential feature is that the corpus luteum should move with the ovary, where an ectopic will move separate from the ovary (you can push the ectopic away from it). Also, the tubal ring of an ectopic pregnancy is usually more echogenic when compared to the ovarian parenchyma. Whereas, the wall of the corpus luteum is usually less echogenic. A specific (but not sensitive) finding in ectopic pregnancy is a RI of <0.4 or >0.7 .

Ectopic	Corpus Luteum
RI < 0.4 , or >0.7	RI 0.4-0.7
Thick echogenic rim	Thin Echogenic Rim
“Ring of Fire”	“Ring of Fire”
Moves Separate from the Ovary	Moves with the Ovary

Endometrioma: This affects young women during their reproductive years and can cause chronic pelvic pain associated with menstruation. The traditional clinical history of endometriosis is the triad of infertility, dysmenorrhea, and dyspareunia. The buzzword classic appearance is **rounded mass with homogeneous low level internal echoes and increased through transmission (seen in 95% of cases)**. Fluid-fluid levels and internal septations can also be seen. As mentioned above it can look a lot like a hemorrhagic cyst (sometimes). As a general rule, the more unusual or varied the echogenicity and the more ovoid or irregular the shape, the more likely the mass is an endometrioma. Additionally, and of more practical value, they are not going to change on follow up (hemorrhagic cysts are). In about 30% of cases you can get small echogenic foci adhering to the walls (this helps make the endometrioma diagnosis more likely). Obviously, you want to differentiate this from a true wall nodule. The complications of endometriosis (bowel obstruction, infertility, etc...) are due to a fibrotic reaction associated with the implant.

Do Endometriomas Ever Become Cancer? About 1% of endometriomas undergo malignant transformation (usually endometrioid or **clear cell carcinoma**). How do you tell which one is which??? Malignancy is very rare in endometriomas smaller than 6 cm. They usually have to be bigger than 9 cm. Additionally, the majority of women with carcinoma in an endometrioma are older than 45 years. So **risk factors for turning into cancer: (a) older than 45, (b) bigger than 6-9cm.**

Endometrioma on MRI: Will be T1 bright (from the blood). Fat saturation will not suppress the signal (showing you it's not a teratoma). Will be T2 dark! (from iron in the endometrioma). The shading sign is a buzzword for endometriomas on MR imaging. On T2 you should look for "shading." **The shading sign, describes T2 shortening (getting dark) of a lesion that is T1 bright.**



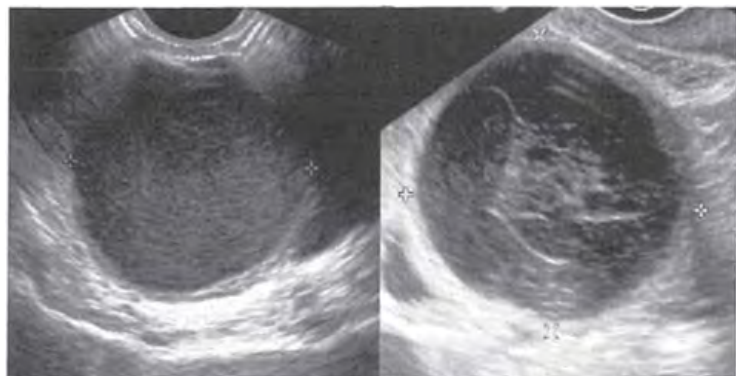
- Minimal Peripheral enhancement is ok.
- Solid enhancing components are worrisome for malignancy.

Endometrioma - "Shading Sign" - Darker on T2

Hemorrhagic Cysts: As mentioned above, sometimes a ruptured follicle bleeds internally and re-expands. The result is a homogenous mass with **enhanced through transmission** (*tumor won't do that*) with a very similar look to an endometrioma. A lacy "**fishnet appearance**" is sometimes seen and is considered classic. Doppler flow will be absent. The traditional way to tell the difference between a hemorrhagic cyst vs endometrioma, is that the **hemorrhagic cyst will go away in 1-2 menstrual cycles** (so repeat in 6-12 weeks).

Hemorrhagic Cyst on MRI - Will be T1 bright (from the blood). Fat saturation will not suppress the signal (showing you it's not a teratoma). The lesion should NOT enhance.

Hemorrhagic cysts in old ladies? Postmenopausal women may occasionally ovulate, so you don't necessarily need to freak out (follow up in 6-12 weeks). Now, late postmenopausal women should NEVER have a hemorrhagic cyst and if you are shown something that looks like a hemorrhagic cyst in a 70 year old - it's cancer till proven otherwise.



Endometrioma
- Homogenous low level echoes - Lacy Fishnet appearance

Dermoid: These things typically occur in young women (20s-30s), and are the most common ovarian neoplasm in patients younger than 20. The “Tip of the Iceberg Sign” is a classic buzzword and refers to absorption of most of the US beam at the top of the mass. The typical ultrasound appearance is that of a cystic mass, with a hyperechoic solid mural nodule, (Rokitansky nodule or dermoid plug). Septations are seen in about 10%.

Dermoid on MRI: Will be bright on T1 (from the fat). There will be fat suppression (not true of hemorrhagic cysts, and endometriomas).



Do Dermoids Ever Become Cancer? About 1% of dermoids can undergo malignant transformation (**almost always to squamous cell CA**). Again, risk factors are size (usually larger than 10cm), and age (usually older than 50).

Rare Cancer Transformation Subtypes

Endometrioma Clear Cell

Dermoid Squamous

Polycystic Ovarian Syndrome: Typically an overweight girl with infertility, acne, and hirsutism.

The imaging criteria is:

- **Ten or more peripheral simple cysts** (*typically small <5mm*)
- Usually Characteristic 'string-of-pearls' appearance.
- **Ovaries are typically enlarged (>10cc)**, although in 30% of patients the ovaries have a normal volume.

Ovarian Cancer

Ovarian cancers often present as complex cystic and solid masses. They are typically intra-ovarian (most extra-ovarian masses are benign). The role of imaging is not to come down hard on histology (although the CORE may ask this of you), but instead to distinguish benign from malignant and let the surgeon handle it from there.

Think Cancer if:

- * Unilateral (or bilateral) complex cystic adnexal masses with thick (>3mm) septations, and papillary projections (nodule with blood flow).
- * Solid adnexal masses with variable necrosis

Knee Jerks:

- * Multiple thin or thick septations = Call the Surgeon
- * Nodule with Flow = Call the Surgeon
- * Solid Nodules Without Flow =
 - o Get an MR to make sure it's not a dermoid plug,
 - o Not a dermoid, then call the surgeon

Serous Ovarian / Cystadenocarcinoma / Cystadenoma

Serous tumors are the **most common type of ovarian malignancy**. About 60% of serous tumors are benign, and about 15% are considered borderline (the rest are malignant). They favor women of childbearing age, with the malignant ones tending to occur in older women. They typically are unilocular with few septations. They are frequently bilateral (especially when malignant). Papillary projections are a common finding, which suggests malignancy. If you see ascites they have mets (70% have peritoneal involvement at the time of diagnosis).



Serous Ovarian Neoplasm
- Large, Unilocular, Few Septations

Mucinous Ovarian Cystadenocarcinoma

Often a large (**can be monster large**) mass. They are **typically multiloculated** (although septa are often thin). **Papillary' projections are less common** with serous tumors. You can see low level echos (from mucin). These dudes can get Pseudomyxoma peritonei with scalloping along solid organs. Smoking is a known risk factor (especially for mucinous types).

Endometrioid Ovarian Cancer: This is the second most common ovarian cancer (serous number one, mucinous number three). These things are bilateral about 15% of the time.

What to know:

- *Associated with endometrial cancer (ovarian mass + endometrial thickening)*
- *Endometriomas can turn into endometrioid cancer*

Fibroma / Fibrothecoma:

The ovarian fibroma is a benign ovarian tumor, most commonly seen in middle aged women. The fibrothecoma / thecoma spectrum has similar histology. It's **very similar to a fibroid**. On ultrasound it's going to be hypoechoic and solid. On MR1 its going to be T1 and T2 dark, with the **band of T2 dark signal around the tumor on all planes**. **Calcifications are rare**

Similar or Related Conditions:

- **Meigs Syndrome:** This is the triad of ascites, pleural effusion, and a benign ovarian tumor (most commonly fibroma).



- **Fibromatosis:** This is a zebra. You have tumor-like enlargement of the ovaries due to ovarian fibrosis. It typically hits girls around the age of 25. It's associated with omental fibrosis, and sclerosing peritonitis. You are going to get dark T1 and T2 signal. **The buzzword for that T2 signal is "black garland sign."** The condition is benign, and sometimes managed with surgical removal of the ovaries. •

- **Brenner Tumor:** Epithelial tumor of the ovary' seen in women in their 50s-70s. It's fibrous and T2 dark. Unlike Fibromas, calcifications are common (80%).

Metastatic Disease to the Ovary

Around 10% of malignant ovarian tumors are mets. The primary is most common from colon, gastric, breast, lung, and contralateral ovary. The most common look is bilateral solid tumors.

Krukenburg Tumor - This is a metastatic tumor to the ovaries from the G1 tract (usually stomach).

Miscellaneous Topics:

Ovarian Torsion

Rotation of the ovarian vascular pedicle (partial or complete) can result in obstruction to venous outflow and arterial inflow. Torsion is typically associated with a cyst or tumor (any thing that makes it heavy, so it flops over on itself). Critical point = the most constant finding in ovarian torsion is a large ovary.

Features:

- *Unilateral enlarged ovary (greater than 4cm)*
- ***Mass on the ovary***
- *Peripheral Cysts*
- *Free Fluid*
- *Lack of arterial or venous flow*

The Ovary is Not a Testicle: The ovary has a dual blood supply. Just because you have flow, does NOT mean there isn't a torsion. You can torse and de-torse. In other words, big ovary + pain = torsion. *Clinical correlation recommended.*

Hydrosalpinx

Thin (or thick in chronic states) elongated tubular structure in the pelvis. The buzzword is **“cogwheel appearance”**, referring to the normal longitudinal folds of a fallopian tube becoming thickened. Another buzzword is **“string sign”** referring to the incomplete septae. The **“waist sign”** describes a tubular mass with indentations of its opposing walls (this is suppose to help differentiate hydrosalpinx from an ovarian mass).

There are a variety of causes, the most common is being a skank, infidel, or free spirit (PID). Additional causes include endometriosis, tubal cancer, post hysterectomy (without oophorectomy), and tubal ligation.

Rare and late complication is tubal torsion.

Pelvic Inflammatory Disease (PID)

Infection or inflammation of the upper female genital tract. It's usually secondary to skank like behavior (Gonorrhea, or Chlamydia). On ultrasound you are gonna get Hydrosalpinx. The margin of the uterus may become ill defined (**“indefinite uterus”** - is a buzzword). Later on you can end up with tubo-ovarian abscess or pelvic abscess. You can even get bowel or urinary tract inflammatory changes.

Paraovarian Cyst

This is a congenital remnant that arises from the Wolffian duct. They are more common than you think with some texts claiming these account for 10-20% of adnexal masses. They are classically round or oval, simple in appearance, and **do NOT distort the adjacent ovary** (key finding). They can indent the ovary and mimic an exophytic cyst, but a good sonographer can use the transducer to separate the two structures.

Ovarian Vein Thrombophlebitis

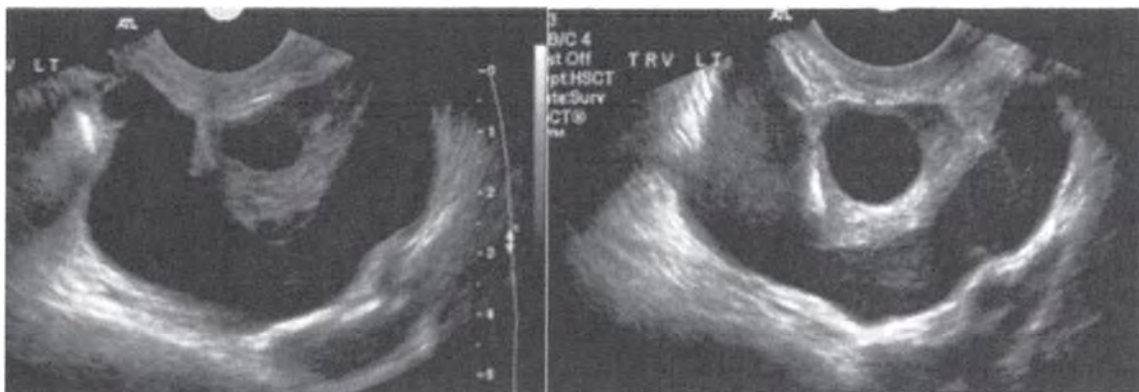
This is seen most commonly in **post partum women**, often presenting with acute pelvic pain and fever. For whatever reason, **80% of the time it's on the right**. It's most likely to be shown on CT (could be ultrasound) with a tubular structure with an enhancing wall and low-attenuation thrombus in the expected location of the ovarian vein. A dreaded sequella is pulmonary embolus.

Peritoneal Inclusion Cyst

This is an inflammatory cyst of the peritoneal cavity, that occurs when adhesions envelop an ovary. Adhesions can be thought of as diseased peritoneum. Whereas the normal peritoneum can absorb fluid, adhesions cannot. So, you end up with normal secretions from an active ovary confined by adhesions and resulting in an expanding pelvic mass. The classic history is patient with prior pelvic surgery (they have to tell you that, to clue you in on the presence of adhesions), now with pain. Alternatively, they could get tricky and say history of PID or endometriosis (some kind of inflammatory process to piss off the peritoneum). Then they will show an ultrasound (or MR) with a complex fluid collection occupying pelvic recesses and containing the ovary. It's not uncommon to have septations, loculations and particulate matter within the contained fluid.

Key features:

- (1) *Lack of walls. They have a “passive shape ” that conforms to and is defined by surrounding structures*
- (2) *Entrapment of an ovary. Ovary will be either in the collection, or at the periphery.*



Peritoneal Inclusion Cyst-Adhesions around an ovary

Gestational Trophoblastic Disease

Think about this with marked elevation of B-hCG. They will actually trend betas for tumor activity. Apparently, elevated B-hCG makes you vomit - so hyperemesis is often part of the given history. Another piece of trivia is that age over 40, and prior moles makes you more likely to get another mole.

Hydatidiform Mole

This is the most common form, and the benign form of the disease. There are type subtypes:

- *Complete mole* (classic mole) (70%): This one involves the entire placenta. There will be no fetus. The worthless trivia is that the karyotype is diploid. A total zebra scenario is that you have a normal fetus, with a complete mole twin pregnancy (if you see that in the wild, write it up). The pathogenesis is fertilization of an egg that has lost its chromosomes (46XX).
 - o *First Trimester US*: Classically shows the uterus to be filled with an echogenic, solid, highly vascular mass, often described as “**snowstorm**” in appearance.
 - o *Second Trimester US*: Vesicles that make up the mole enlarge into individual cysts (2-30mm) and produce your “**bunch of grapes**” appearance.
- *Partial mole* (30%): This one involves only a portion of the placenta. You do have a fetus, but it’s all jacked up (triploid in karyotype). The pathogenesis is fertilization of an ovum by two sperm (69XXY). Mercifully, it’s lethal to the fetus.
 - o *US*: The placenta will be enlarged, and have areas of multiple, diffuse anechoic lesions. You may see fetal parts.

Remember that **I** mentioned that **Theca Lutein cysts are seen in molar pregnancies**. The piece of trivia is that they are actually most commonly seen in the second trimester, and are bilateral.

Invasive Mole

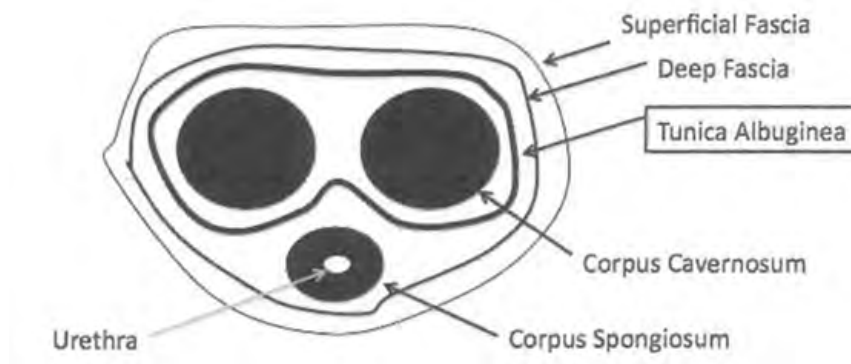
This refers to invasion of molar tissue into the myometrium. You typically see it after the treatment of a hydatidiform mole (about 10% of cases). US may show echogenic tissue in the myometrium. However, MR1 is way better at demonstrating muscle invasive. MRI is going to demonstrate focal myometrial masses, dilated vessels, and areas of hemorrhage and necrosis.

Choriocarcinoma

This is a very aggressive malignancy that forms only trophoblasts (no villous structure). The typical attacking pattern of choriocarcinoma is to spread locally (into the myometrium and parametrium) then to spread hematogenously to any site in the body. It's very vascular and bleeds like stink. The classic clinical scenario is serum P-hCG levels that rise in the 8 to 10 weeks following evacuation of molar pregnancy. On ultrasound, choriocarcinoma (at any site) results in a highly echogenic solid mass. Treatment = methotrexate.

Penis:

Anatomy of this thing in cross section is a high yield topic:



Fractured Penis: This is one of the most tragic situations that can occur in medicine. They can show it on ultrasound (look for hematoma) or MRI (look for hematoma). The piece of trivia you should remember is this is **defined by fracture corpus cavernosum and its surrounding sheath, the tunica albuginea.**

Prostate

Cancer: Biopsy of the prostate is a terrible terrible situation, worse than anything you can imagine in a 1000 years of hell. As a result, MRI of the prostate (instead of biopsy) is getting to be a hot topic. You can use prostate MRI for high risk screening (high or rising PSA with negative biopsy), or to stage (look from extracapsular extension).

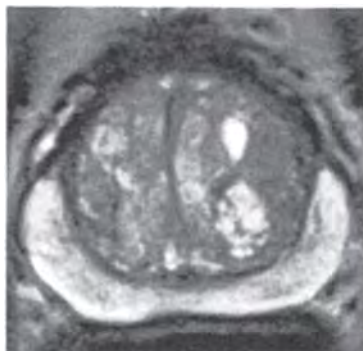
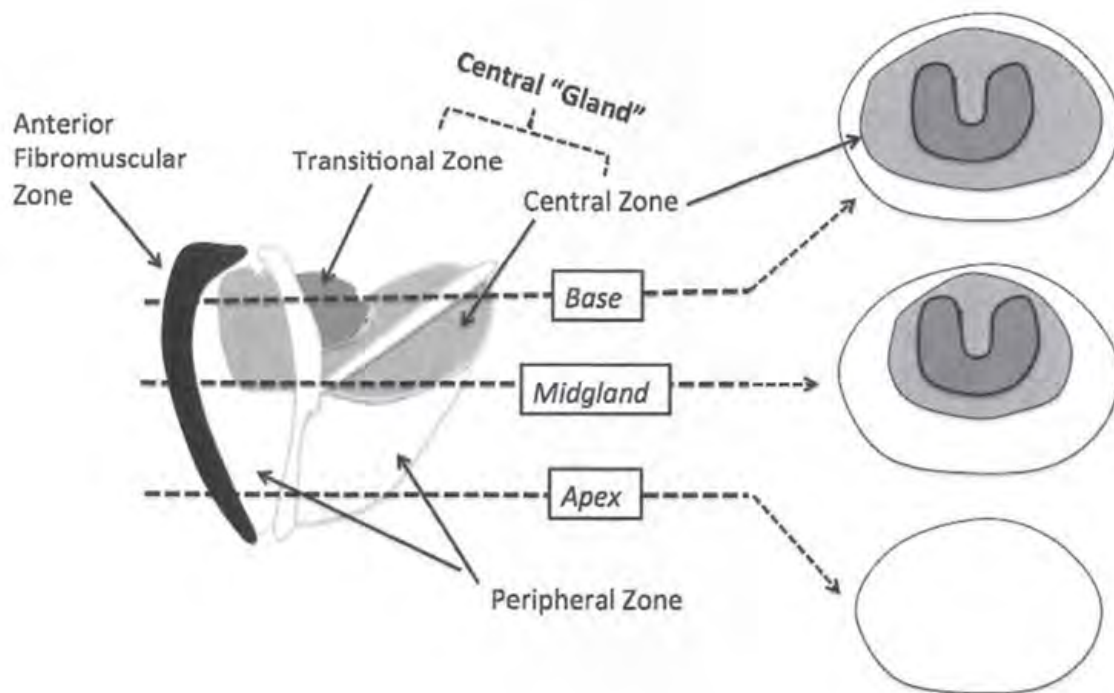
First lets talk about prostate anatomy: Anatomists like to use “zones” to describe locations, and it actually helps with pathology. The anterior fibromuscular gland is dark on T1 and T2. The central and transitional zones (together called the “*central gland*”) are brighter than the anterior muscular zone, but less bright than the peripheral zone on T2. In other words the **peripheral zone is the most T2 bright**.

Adenocarcinoma:

Peripheral Zone: 70%

Transition Zone: 20%

Central Zone: 10 %



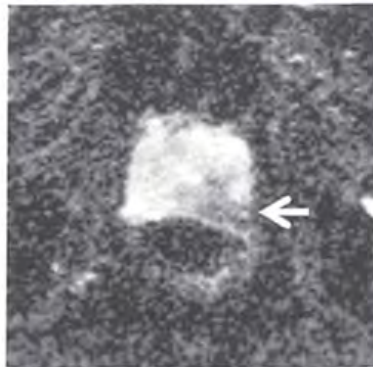
Darker Stuff = Central Gland
(this is where BPH nodules live)

Bright Stuff = Peripheral Zone
(this is where the cancer lives)

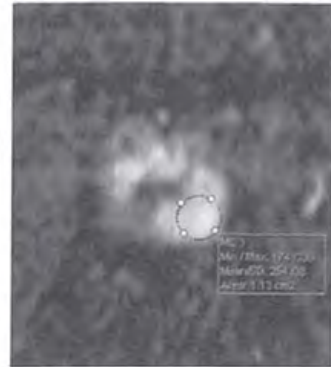
*MRI finding for Prostate CA: **Cancer is dark on T2** (background is high), restricts on diffusion (low on ADC), and enhances early and washes out (type 3 curve - just like a breast cancer).*



Dark on T2



Dark on ADC
(restricts diffusion)



Enhances

Bone scan is the money for prostate mets (vertebral body mets).

Staging: The main thing to know is stage B vs stage C.

.Stage B	Stage C
Confined by capsule	Extension through capsule
Abutment of the capsule without bulging	Bulging of the capsule, or frank extension through it

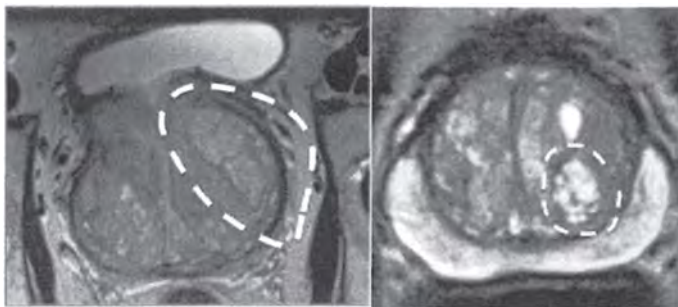
Seminal vesicles and the nerve bundle are also right behind the prostate and can get invaded (urologists love to hear about that).

BPH: Obviously this is super common, and makes old men pee a lot. Volume of 30cc is one definition. **Most commonly involves the transitional zone** (cancer is rare in the transition zone - 10%). The **median lobe** is the one that hypertrophies and sticks up into the bladder. It can cause outlet obstruction, bladder wall thickening (detrusor hypertrophy), and development of diverticulum.

The IVP buzzword is “J shaped” or “Fishhook” shaped ureter.

With regard to the BPH nodules you see on MR1, they are usually:

- In the transitional Zone
- T2 Heterogenous
- Can Restrict Diffusion
- May enhance and washout



BPH Nodules
- *Bright/Heterogeneous*
Shit in the Central Gland

Post Biopsy Changes: This is going to be T1 bright stuff in the gland. It’s subacute blood.

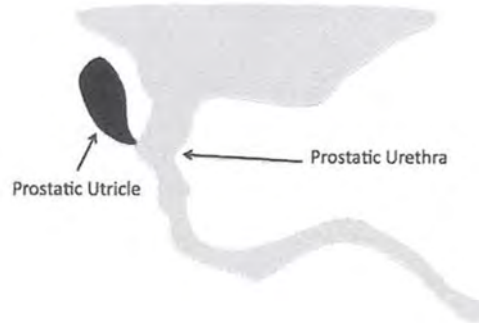
Prostate Summary Chart

	T2	ADC	Enhancement
Peripheral Zone Tumor	Dark	Dark	Early Enhancement, Early Washout
Peripheral Zone Hemorrhage <i>*Like after a biopsy</i>	Dark (sometimes T1 bright)	Dark (less dark)	None
Central Gland / Transitional Tone Tumor	Dark "Charcoal"	Dark	Early Enhancement, Early Washout
BPH	Dark "Well Defined"	Less Dark	Can enhance

Misc Male Reproductive Conditions

Prostatic Utricle Cyst / Mullerian Duct Cyst:

Some people try and distinguish between a prostatic utricle, and a mullerian duct cyst. They look very very similar. The Mullerian duct cyst is an anatomic variant of the caudal ends of the mullerian ducts (male equivalent of the vagina / cervix). The prostatic utricle is a focal dilation in the prostatic urethra. This can be shown with multiple imaging modalities. Think about it if you see a **midline cystic structure near the bladder of a man**. A sneaky trick would be to show it on a RUG, where a prostate utricle cyst would look like a focal outpouching from the prostatic urethra.



Things to know (prostatic utricle cyst):

- *Hypospadias is the most common associated condition*
- *Prune Belly Syndrome, Downs, unilateral renal agenesis, and Imperforate Anus are also associated*
- *If its large, it can get infected*

Things to know (mullerian duct cyst)

- *Does not have the same associations as utricle cyst*
- *Can contain cancer (various types: endometrial, clear cell, squamous).*

Seminal Vesicle Cysts: The classic look is a **unilateral, lateral cyst** (lateral to the prostate). If they get large they can look midline, but if they show you a large one you won't be able to tell it from the utricle cyst. They can be congenital or acquired.

Congenital Trivia:

- *Associated with renal agenesis*
- *Associated with vas deferens agenesis*
- *Associated with ectopic ureter insertion*

Acquired Trivia:

- *Obstruction often from prostatic hypertrophy, or chronic infection/scarring*
- *Classic history is prior prostate surgery*

Male Pelvic Cysts	
<i>Midline</i>	<i>Lateral</i>
Prostatic Utricle	Seminal Vesicle Cyst
Mullerian Duct Cyst	Diverticulosis of the ampulla of vas deferens
Ejaculatory Duct Cysts	

Testicular Trauma: Surgical intervention is required if there is testicular rupture. Intratesticular fracture, and hematomas (small) do not get surgery.

- Rupture:** Disrupted tunica albuginia, heterogenous testicle, poorly defined testicular outline
- Fracture:** Intact tunica albuginia, linear hypoechoic band across the parenchyma of the testicle, well defined testicular outline.

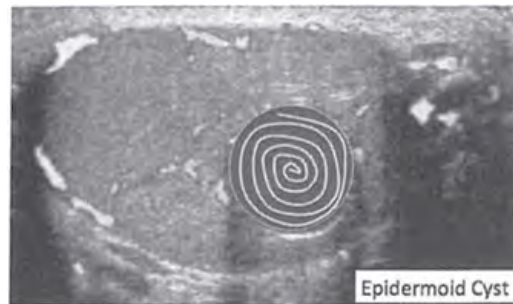
Torsion of the Testicle - Results from the testis and spermatic cord twisting within the serosal space leading to ischemia. If it was 1950 you'd call in your nuclear medicine tech for scintigraphy. Now you just get a Doppler ultrasound. Findings will be absent or asymmetrically decreased flow, asymmetric enlargement and slightly decreased echogenicity of the affected testis.

- Cause:** The "bell-clapper deformity" which describes an abnormal high attachment of the tunical vaginalis, increases mobility and predisposes to torsion. It is usually a bilateral finding, so the contralateral side gets an orchiopexy.
- Viability:** The viability is related to the degree of torsion (how many spins), and how long it has been spun. As a general rule, the surgeons try and get them in the OR before 6 hours.

Epididymitis - Inflammation of the epididymis, and the most common cause of acute onset scrotal pain in adults. In sexually active men the typical cause is chlamydia or gonorrhea. In older men its more likely to be e-coli, due to a urinary tract source. The epididymal head is the most affected. Increased size and hyperemia are your ultrasound findings. You can have infection of the epididymis alone or infection of the epididymis and testicle (isolated orchitis is rare).

Orchitis - Typically progresses from epididymitis (isolated basically only occurs from mumps). It looks like asymmetric hyperemia.

Epidermoid Cysts: This is a benign mass of the testicle (no malignant potential), with an Aunt Minnie “**onion skin**” look, - alternating hypoechoic and hyperechoic rings. It’s relatively non-vascular relative to the rest of the testicle.



Tubular Ectasia of the Rete Testis: This is a common benign finding, resulting from obliteration (complete or partial) of the efferent ducts. It’s usually bilateral - and in older men. The location of the cystic dilation is next to the mediastinum testis. Think about this as a normal variant. It requires no follow up or further evaluation.



Calcified Vas Deferens: You see this all the time in bad **diabetics**.

Calcified Vas Deferens

Testicular Cancer:

Testicle Cancer in the pediatric setting is discussed in the pediatric chapter. This discussion will focus of the adult subtypes (with some overlap).

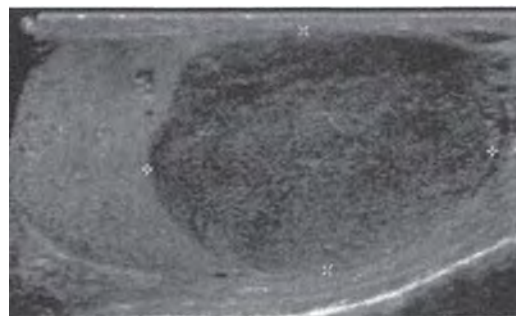
In general, hypoechoic solid intratesticular masses should be thought of as cancer until proven otherwise. Doppler flow can be helpful only when it is absent (can suggest hematoma - in the right clinical setting). If it’s extratesticular and cystic it’s probably benign. The step 1 trivia is that cryptorchidism increases the risk of cancer (in both testicles), and is not reduced by orchiopexy. Most testicular tumors met via the lymphatics (retroperitoneal nodes at the level of the renal hilum). The testable exception is choriocarcinoma, which mets via the blood. Most testicular cancers are germ cell subtypes (95%) - with seminomas making up about half of those.

Risk Factors: Cryptorchidism (for both testicles), Gonadal Dysgenesis, Klinefelters, Trauma, Orchitis, and testicular microlithiasis (maybe).

Testicular Microlithiasis - This appears as multiple small echogenic foci within the testes. Testicular microlithiasis is usually an incidental finding in scrotal US examinations performed for unrelated reasons. It might have a relationship with Germ Cell Tumors (controversial). Follow-up in 6 months, then yearly is probably the recommendation - although this recommendation is controversial.



Seminoma: This is the most common testicular tumor, and has the best prognosis as they are very radiosensitive. They are much more common (9x) in white people. The classic age is around 25. It usually looks like a homogenous hypoechoic round mass, which classically replaces the entire testicle. On MRI they are usually homogeneously T2 dark (non-seminomatous GCTs are often higher in signal).



Seminoma

Non-Seminomatous Germ Cell Tumors: Basically this is not a seminoma. We are talking about mixed germ cell tumors, teratomas, yolk sac tumors, and choriocarcinoma. They typically occur at a young age relative to seminomas (think teenager). They are more heterogenous and have larger calcifications.

Testicular Lymphoma - Just be aware that lymphoma can “hide” in the testes because of the blood testes barrier. Immunosuppressed patients are at increased risk for developing extranodal/ testicular lymphoma. Almost all testicular lymphomas are non-hodgkin B-cell subtypes. On US, the normal homogeneous echogenic testicular tissue is replaced focally or diffusely with hypoechoic vascular lymphomatous tissue **Buzzword = multiple hypoechoic masses of the testicle.**

Burned Out Testicular Tumor - If you see large dense calcifications with shadowing in the testicle of an old man this is probably what you should be thinking. The idea is that you’ve had spontaneous regression of a genu cell testicular neoplasm, that is now calcified. An important pearl is that there can still be viable tumor in there. Management is somewhat controversial and unlikely to be asked (most people pull them out).

High Yield Testicle Tumor Trivia

Seminoma is the most common, and has the best prognosis (it melts with radiation)

Multiple hypoechoic masses = Lymphoma

Homogenous and Microcalcifications = Seminoma

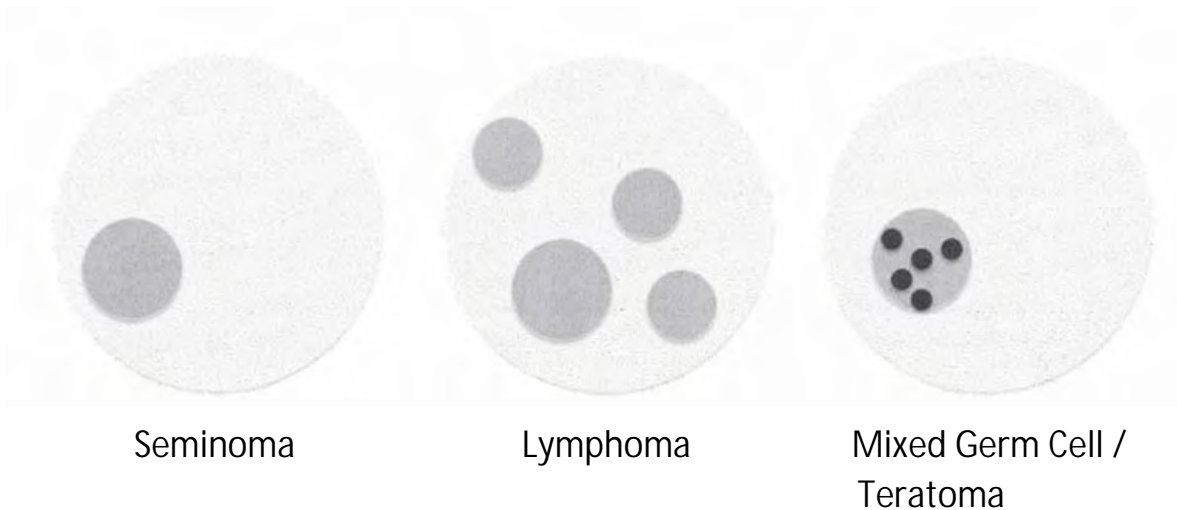
Cystic Elements and Macrocalcifications = Mixed Germ Cell Tumor/ Teratoma

Most testicular tumors met via the lymphatics (choriocarcinoma mets via the blood - and tends to bleed like stink)

Gynecomastia can be seen with Sertoli Leydig Tumors

Sertoli Cell Tumors are also seen with Peutz Jeghers

Elevated hCG	Elevated AFP
Seminoma	Mixed Germ Cell
Choriocarcinoma	Yolk Sac



Male Infertility

Causes: Can be thought of as obstructive vs non obstructive.

- * Obstructive: Congenital bilateral absence of the vas deferens (seen in Cystic Fibrosis), ejaculatory duct obstruction, prostatic cysts. **Think about associated renal anomalies (Zinner Syndrome).**
- Non-Obstructive: Varicocele, Cryptorchidism, Anabolic Steroid Use, Erectile Dysfunction.

Varicocele: This is **the most common correctable cause of infertility**. They can be unilateral or bilateral. Unilateral is much more common on the left. Isolated **right sided** **should make you think retroperitoneal process** compressing the right gonadal vein.

Cryptorchidism: Undescended testes. The testicle is usually found in the inguinal canal. The testicle has an increased risk of cancer (actually they both will - which is weird). It's **most commonly seen in premature kids (20%)**.



Zebras and Syndromes associated with male infertility:

- * Pituitary Adenoma making prolactin
- * Kallmans Syndrome (can't smell + infertile)
- * Klinefelters Syndrome (tall + gynecomastia + infertile)
- * Zinner Syndrome (renal agenesis + ipsilateral seminal vesicle cyst)

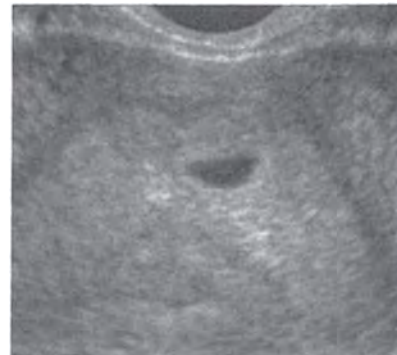
OB

Early Pregnancy:

Vocab:

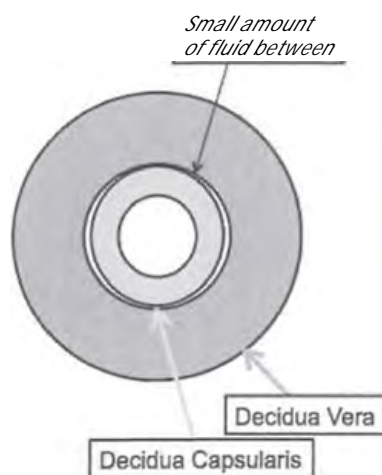
- Menstrual Age: Embryologic Age + 14 days
- Embryo: 0-10 weeks (menstrual age)
- Fetus: > 10 weeks (menstrual age)
- Threatened Abortion - Bleeding with closed cervix
- Inevitable Abortion - Cervical dilation and/or placental and/or fetal tissue hanging out
- Incomplete Abortion - Residual products in the uterus
- Complete Abortion - All products out
- Missed Abortion - Fetus is dead, but still in the uterus.

Intradecidual Sign: This is the early gestational sac. When seen covered by echogenic decidua is very characteristic of early pregnancy. You can see it around 4.5 weeks. You want to see the thin echogenic line of the uterine cavity pass by (not stop at) the sac to avoid calling a little bit of fluid in the canal a sac.



Intradecidual Sign

Double Decidual Sac Sign: This is another positive sign of early pregnancy. It's produced by visualizing the layers of decidua.



Double Decidual Sac Sign

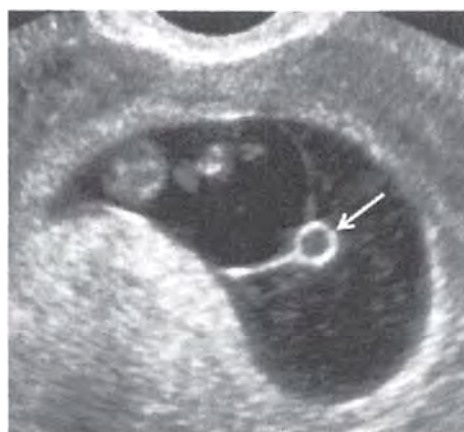
Yolk Sac: This is the first structure visible within the GS. The classic teaching was you should always see it when the GS measures 8mm in diameter. The thing should be oval or round, fluid filled, and smaller than 6mm.

The yolk sac is located in the chorionic cavity, and hooked up to the umbilicus of the embryo by the vitelline duct.

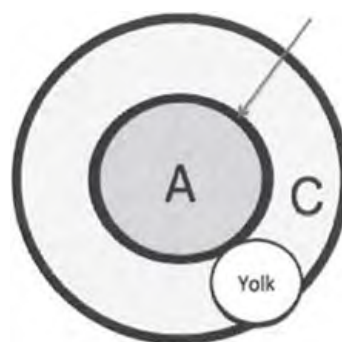
Yolk Sac Gone Bad: The yolk sac shouldn't be too big ($> 6\text{mm}$), shouldn't be too small ($< 3\text{mm}$), and shouldn't be solid or calcified.

The Amnion: The membranes of the amniotic sac and chorionic space typically remain separated by a thin layer of fluid, until about 14-16 weeks at which point fusion is normal. If the amnion gets disrupted before 10 weeks the fetus might cross into the chorionic cavity and get tangled up in the fibrous bands. This is the etiology of **amniotic band syndrome**, which can be terrible (decapitation, limb amputation, etc...).

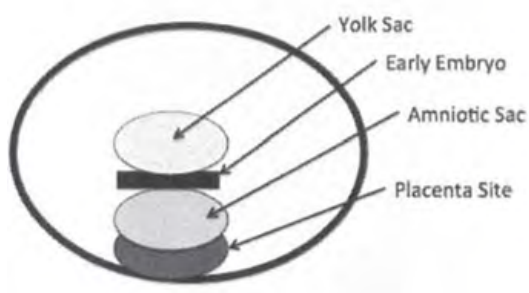
Double Bleb Sign: This is the earliest visualization of the embryo. This is two fluid filled sacs (yolk and amniotic) with the flat embryo in the middle.



Yolk Sac – in the chorionic cavity



A = Amniotic Space
Arrow = Amnion
C = Chorionic Space

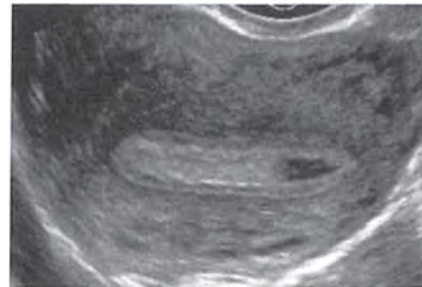


Double Bleb Sign

Crown Rump Length - This is typically used to estimate gestational age, and is more accurate than menstrual history.

Anembryonic Pregnancy - A gestational sac without an embryo. When you see this, the choices are (a) very early pregnancy, or (b) non-viable pregnancy. The classic teaching was you should see the yolk sac at 8mm (on TV). Just remember that a large sac (>8-10 mm) without a yolk sac, and a distorted contour is pretty reliable for a non-viable pregnancy.

Pseudogestational Sac - This is not the same thing as an anembryonic pregnancy. This is seen in the presence of an ectopic pregnancy. What you are seeing is a little bit of blood in the uterine cavity with surrounding bright decidual endometrium (charged up from the pregnancy hormones).



Pseudogestational Sac

Old criteria for fetal demise vs new criteria for

fetal demise - They have recently (very recently) redone the numbers for calling it non-viable and I'm not sure which version will be tested so I'm presenting both. For the most part, the new ones give more wiggle room.

Most Recent Guideline for Fetal Demise (maybe too new for CORE)		Old School (Traditional) Guidelines for Abnormal Pregnancy
Diagnostic of Pregnancy Failure	Suspicious for Pregnancy Failure	
Crown-rump length of >7 mm and no heartbeat	No embryo >6 wk after last menstrual period	Absent Yolk Sac with MSD > 8mm
Mean sac diameter of >25 mm and no embryo	Mean sac diameter of 16-24 mm and no embryo	Absent Embryo when MSD > 16mm
No embryo with heartbeat >11 days after a scan that showed a gestational sac with a yolk sac	No embryo with heartbeat 13 days after a scan that showed a gestational sac without a yolk sac	No Cardiac Activity when Embryo can be seen on Transabdominal (or >5mm on TV). "5 Alive". ** Always give 1-2mm as the benefit of doubt.
No embryo with heartbeat >2 wk after a scan that showed a gestational sac without a yolk sac	No embryo with heartbeat 10 days after a scan that showed a gestational sac with a yolk sac	

Doubilet, Peter M., et al. "Diagnostic criteria for nonviable pregnancy early in the first trimester" New England Journal of Medicine 369.15 (2013): 1443-1451.

Subchorionic Hemorrhage: These are very common. The thing to know is that the percentage of placental detachment is the prognostic factor most strongly associated with a fetal demise; hematoma greater than 2/3 the circumference of the chorion has a 2x increased risk of abortion. Other trivia, women older than 35 have worse outcomes with these.

Implantation Bleeding: This is a nonspecific term referring to a small subchorionic hemorrhage that occurs at the attachment of the chorion to the endometrium.

Ectopic:

High Risk for Ectopic: Hx of PID, Tubal Surgery, Endometriosis, Ovulation Induction, Previous Ectopic, Use of an IUD.

The majority of ectopic pregnancies (nearly 95%) occur in the fallopian (usually the isthmic portion). A small percentage (around 2%) are “interstitial” developing in the portion of the tube which passes through the uterine wall. These interstitials are high risk, as they can grow large before rupture causing a catastrophic hemorrhage. It is also possible (although very rare) to have implantation sites in the abdominal cavity, ovary, and cervix.

Always start down the ectopic pathway with a positive BhCG. At around 2000 IU/L you should see a gestational sac. As a general rule, a normal doubling time makes ectopic less likely.

The Big 3 to Remember with Ectopics (positive BhCG)

- (1) Live Pregnancy / Yolk Sac outside the uterus = Slam Dunk
- (2) Nothing in the uterus + anything on the adnexa (other than corpus luteum) = 75-85% PPV for ectopic
 - a. A moderate volume of free fluid increases this to 97% PPV
- (3) Nothing in the uterus + moderate free fluid = 70% PPV
 - a. More risk if the fluid is echogenic

Tubal Ring Sign: An echogenic ring, which surrounds an unruptured ectopic pregnancy. This is an excellent sign of ectopic pregnancy - and has been described as 95% specific.



Tubal Ring Sign

Heterotopic Pregnancy: This is a baby in the uterus and a baby in the tube (or other ectopic location). This is pretty rare, and typically only seen in women taking ovulation drugs, or prior bad PID.

Fetal Biometry and Fetal Growth:

In the second and third trimesters, four standard measurements of fetal growth are made (Biparietal, Head Circumference, Abdominal Circumference, and Femur Length). The testable trivia seems to include what level you make the measurement, and what is and is not included (see chart).

Fetal Measurement For Growth			
	Measurement Made	NOT including	Trivia
Biparietal Diameter “BPD”	Recorded at the level of the thalamus from the outermost edge of the near skull to the inner table of the far skull		affected by the shape of the fetal skull (false large from brachycephaly, false small from dolichocephaly)
Head Circumference	Recorded at the same slice as BPD	Does NOT include the skin	affected less by head shape
Abdominal Circumference	Recorded at the level of the junction of the umbilical vein and left portal vein	Does NOT include the subcutaneous soft tissues	
Femur Length	Longest dimension of the femoral shaft	Femoral epiphysis is NOT included	

Estimated Fetal Weight: This is calculated by the machine based on either (1) BPD and AC, or (2) AC and FL.

Gestational Age (GA): Ultrasound estimates of gestation age are the most accurate in an early pregnancy (and become less precise in the later portions). Age in the first trimester is made from crown rump length. Second and third trimester estimates for age are typically done using BPD, HC, AC, and FL - and referred to as a “composite GA.”

<i>Gestation Age (Less good later in the pregnancy)</i>	
First Trimester - Crown Rump Length	Accurate to 0.5 weeks
2 nd and 3 rd Trimester - “Composite GA”	Accurate to 1.2 weeks (between 12 and 18 weeks)
	Accurate to 3.1 weeks (between 36 and 42 weeks)

Intrauterine Growth Retardation:

Readings Suggestive of IUGR:

- * Estimated Fetal Weights Below 10th percentile
- * Femur Length / Abdominal Circumference Ratio (F / AC) > 23.5
- * Umbilical Artery Systolic / Diastolic Ratio > 4.0

Not All is lost: If the kid is measuring small, he might just be a little guy. If he has normal Doppler studies - most of the time they are ok.

Maybe All is lost: If the kid is measuring small, suggesting IUGR and he has oligohydramnios (AFI < 5) or polyhydramnios he/she is probably toast.

Symmetrical vs Asymmetrical:

- *Asymmetrical:* Think about this as a restriction of weight followed by length. It is the more common of the two types. The head will be normal in size, with the body being small. Some people call this “**head sparing,**” as the body tries to protect the brain. You see this mainly in the third trimester, as a result of extrinsic factors.

The classic scenario would be normal growth for the first two trimesters, with a normal head / small body in the third trimester - with a mom having chronic high BP / pre-eclampsia.

There are a bunch of causes. I recommend remembering these three: **High BP, Severe Malnutrition, Ehler-Danlos.**

- *Symmetric:* This is a global growth restriction, that **does NOT spare the head.** This is **seen throughout the pregnancy** (including the first trimester). The head and body are both small. This has a **much worse prognosis**, as the brain doesn’t develop normally.

There are also a bunch of causes. I recommend remembering these: **TORCH infection, Fetal Alcohol Syndrome / Drug Abuse, Chromosomal Abnormalities, and Anemia.**

Biophysical Profile: This thing was developed to look for acute and chronic hypoxia. Points are assigned (2 for normal, 0 for abnormal). A score of 8-10 is considered normal. To call something abnormal, technically you have to be watching for 30 mins.

Components of Biophysical Profile		
Amniotic Fluid	At least one pocket that measures 2cm or more in a vertical plane	Assess Chronic Hypoxia
Fetal Movement	3 discrete movements	Assess Acute Hypoxia
Fetal Tone	1 episode of fetal extension from flexion	Assess Acute Hypoxia
Fetal Breathing	1 episode of “Breathing motion” lasting 30 seconds	Assess Acute Hypoxia
Non-stress Test	2 or more fetal heart rate accelerations of at least 15 beats per minute and or 30 seconds or longer	Assess Acute Hypoxia

Umbilical Artery Systolic / Diastolic Ratio: The resistance in the umbilical artery should progressively decrease with gestational age. **The general rule is 2-3 at 32 weeks.** The ratio should not be more than 3 at 34 weeks. An elevated S/D ratio means there is high resistance. High resistance patterns are seen in pre-eclampsia and IUGR. Worse than an elevated ratio, is absent or reversed diastolic flow - this is associated with a very poor prognosis.

Macrosomia: Babies that are too big (above the 90th percentile). **Maternal diabetes** (usually gestational, but could be type 2 as well), is the most common cause. As a point of trivia, type 1 diabetic mothers can also have babies that are small secondary to hypoxia from microvascular disease of the placenta. The big issue with being too big is **complications during delivery** (shoulder dystocia, brachial plexus injury) and **after delivery** (neonatal hypoglycemia, meconium aspiration).

Erb's Palsy: Injury to the upper trunk of the brachial plexus (C5-C6), most commonly seen in shoulder dystocia (which kids with macrosomia are higher risk for).



If you see an aplastic or hypoplastic humeral head / glenoid in a kid, you should immediately think about an Erbs Palsy.

Clinical Correlation.

Amniotic Fluid: Early on, the fluid in the amnion and chorionic spaces is the result of filtrate from the membranes. After 16 weeks, the fluid is made by the fetus (urine). The balance of too much (polyhydramnios) and too little (oligohydramnios) is maintained by swallowing of the urine and renal function. In other words, if you have too little fluid you should think kidneys aren't working. If you have too much fluid you should think swallow or other GI problems. Having said that, a common cause of too much fluid is high maternal sugars (gestational diabetes). Fine particulate in the fluid is normal, especially in the third trimester.

Amniotic Fluid Index: Made by measuring the vertical height of the deepest fluid pocket in each quadrant of the uterus then summing the 4 measurements. **Normal is 5-20.**

Oligohydramnios is defined as AFI < 5cm. Polyhydramnios is defined as AFI > 20cm , or a single fluid pocket > 8cm.

Normal Development:

I'm going to briefly touch on what I think is testable trivia regarding normal development.

Brain: Choroid plexus is large and echogenic. There should be less than 3mm of separation of the choroid plexus from the medial wall of the lateral ventricle (if more it's ventriculomegaly). The cisterna magna should be between 2mm-11mm (too small think Chiari II, too large think Dandy Walker).

Face / Neck: The “fulcrum” of the upper lip is normal, and should not be called a cleft lip.

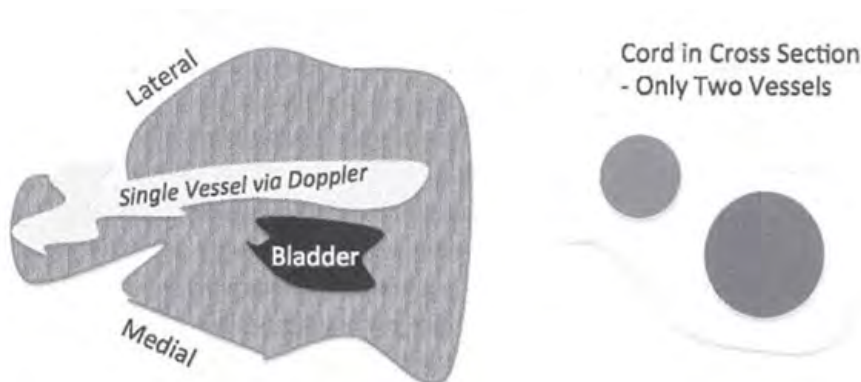
Lungs: The lungs are normally homogeneously echogenic, and similar in appearance to the liver.

Heart: The only thing to know is that a papillary muscle can calcify “Echogenic Foci in the ventricle”, and although this is common and can mean nothing - it's also associated with an increased risk of Downs (look hard for other things).

Abdominal: If you only see one artery adjacent to the bladder, you have yourself a two vessel cord. Bowel should be less than 6mm in diameter. Bowel can be moderately echogenic in the 2nd and 3rd trimester but should never be more than bone. The adrenals are huge in newborns, and are said to be 20x their relative size.

Two Vessel Cord - Gamesmanship

There are two main ways to show a two vessel cord. The first one is a single vessel running lateral to the bladder down by the cord insertion. The second is to show the cord in cross section with two vessels.



More on the 2 vessel cord in a few pages.

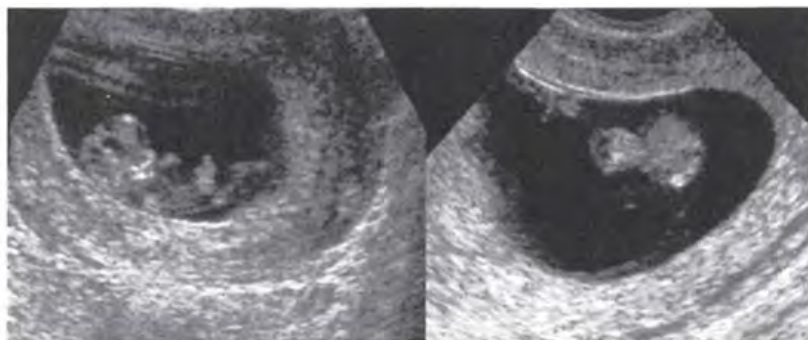
Classic Normal Pictures That Look Scary

- (1) **Cystic Rhombencephalon:** The normal rhombencephalon is present as a cystic structure in the posterior fossa around 6-8 weeks. *Don't call it a Dandy- Walker malformation*, for sure that will be a distractor.



Normal Cystic Rhombencephalon (6-8 weeks)

- (2) **Physiologic Mid Gut Herniation:** The midgut normally herniates into the umbilical cord around 9-11 weeks. *Don't call it an omphalocele*, for sure that will be a distractor.



Normal Midgut Herniation (9-11 weeks)

Placenta and Umbilical Cord

Placenta

Normal: You can first start to see the placenta around 8 weeks (focal thickening along the periphery of the gestational sac). It should be shaped like a disc around 12 weeks. The normal sonographic appearance is “granular” with a smooth cover (the chorion). Underneath the basal surface there is a normal retroplacental complex of decidual and myometrial veins.

Normal Placenta Aging: As the placenta ages it gets hypoechoic areas, septations, and randomly distributed calcifications.

Venous Lakes: These are an incidental finding of no significance. They look like focal hypoechoic areas under the chorionic membrane (or within the placenta). You can sometimes see slow flow in them.

Placental Thickness	
Too Thin (< 1 cm)	Too Thick (> 4cm)
Placental Insufficiency, Maternal Hypertension, Maternal DM, Trisomy 13, Trisomy 18, Toxemia of Pregnancy	Fetal Hydrops, Maternal DM, Severe Maternal Anemia, Congenital Fetal Cancer, Congenital Infection, Placental Abruption

Variant Placental Morphology:

Bilobed Placenta	Two near equal sized lobes	Increased risk of type 2 vasa previa, post partum hemorrhage from retained placental tissue, and velamentous insertion of the cord
Succenturiate Lobe	One or more small accessory lobes	Increased risk of type 2 vasa previa, post partum hemorrhage from retained placental tissue
Circumvallate Placenta	Rolled placental edges with smaller chorionic plate	High risk for placental abruption and IUGR

Placenta Previa: This is a low implantation of the placenta that covers part of or all of the internal cervical os. The clinical buzzword is “painless vaginal bleeding in the third trimester.” A practical pearl is that you need to have an empty bladder when you look for this (full bladder creates a false positive).

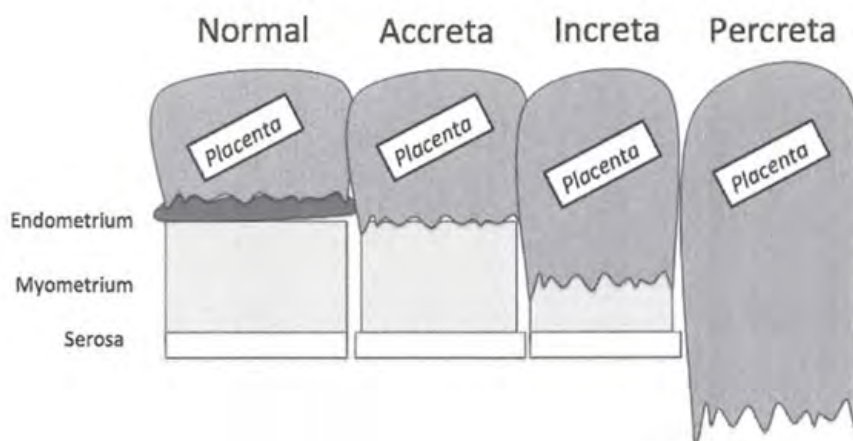
Placental Abruption: This is a premature separation of the placenta from the myometrium. The step 1 history was always “mother doing cocaine”, but it also occurs in the setting of hypertension. Technically subchorionic hemorrhage (marginal abruption) is in the category - as previously discussed. Retroplacental Abruption is the really bad one. The hematoma will appear as anechoic or mixed echogenicity beneath the placenta (often extending beneath the chorion). Buzzword is “disruption of the retroplacental complex.”

Placental Abruption vs Myometrial Contraction / Fibroid	
Placental Abruption will disrupt the retroplacental complex of blood vessels	Myometrial Contractions / Fibroids will displace the retroplacental complex

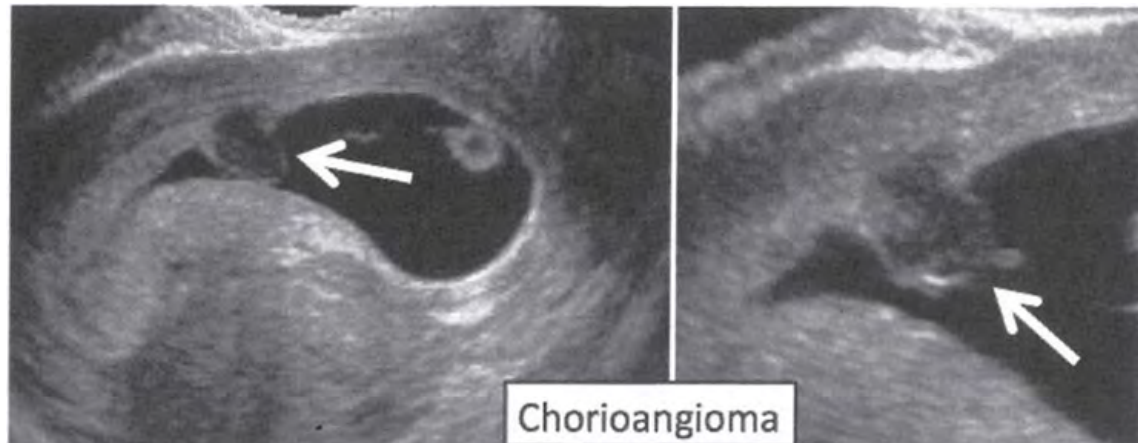
Placenta Creta: This is an abnormal insertion of the placenta, which invades the myometrium. The severity is graded with fancy sounding Latin names. The risk factors include prior C-section, placenta previa, and advanced maternal age. The sonographic appearance varies depending on the severity, but generally speaking you are looking for a “moth-eaten” or “Swiss cheese” appearance of the placenta, with vascular channels extending from the placenta into the myometrium (with turbulent flow on Doppler). Thinning of the myometrium (less than

1 mm) is another sign. This can be serious business, with life threatening bleeding sometimes requiring hysterectomy. The **big risk factor is prior c-section, and placenta previa.**

Placenta Accreta	Most common (75%) and mildest form, the villi attach to the myometrium, without invading.
Placenta Increta	Villi partially invade the myometrium
Placenta Percreta	the really bad one. Villi penetrate through the myometrium or beyond the serosa. Sometimes there is invasion of the bladder or bowel.



Placenta Chorioangioma: This is basically a **hamartoma** of the placenta, and is the most common benign tumor of the placenta. These are usually well circumscribed hypoechoic masses **near the cord insertion**. Flow within the mass pulsating at the fetal heart rate is diagnostic (they are perfused by the fetal circulation). They almost always mean nothing, but if they are large (> 4cm) and multiple (“chorioangiomatosis”) they can sequester platelets, and cause a high output failure (hydrops).



Placental Chorioangioma vs Placental Hematoma	
Chorioangioma has pulsating Doppler flow	Hematoma does NOT have pulsating Doppler flow.

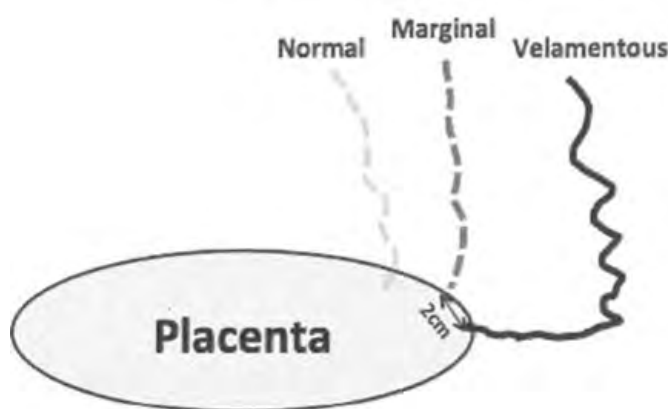
Umbilical Cord

Normal Cord: Should have 3 vessels (2 arteries, 1 vein).

Two Vessel Cord: This is a normal variant - seen in about 1% of pregnancies. Usually the left artery is the one missing. This tends to occur more in twin pregnancies and maternal diabetes. There is an increased association with chromosomal anomalies and various fetal malformations (so look closely). Having said that, in isolation it doesn't mean much.

Velamentous Cord Insertion: This is the term for when the cord inserts into the fetal membranes outside the placental margin, and then has to travel back through the membranes to the placenta (between the amnion and the chorion). It's more common with twins, and increases the risk of intra-uterine growth restriction and growth discordance among twins.

Marginal Cord Insertion: This is basically almost a velamentous insertion (cord is within 2cm of the placental margin). It's also seen more in twin pregnancies.



Vasa Previa: Fetal vessels that cross (or almost cross) the internal cervical os. It's seen more in twin pregnancies, and variant placental morphologies. There are two types:

- Type 1: Fetal vessels connect to a velamentous cord insertion within the main body of the placenta
- Type 2: Fetal vessels connect to a bilobed placenta or succenturiate lobe.

Nuchal Cord: This is the term used to describe a cord wrapped around the neck of the fetus. Obviously this can cause problems during delivery.

Umbilical Cord Cyst: These are common (seen about 3% of the time), and are usually single (but can be multiple). As a point of completely irrelevant trivia you can divide these into false and true cysts. True cysts are less common, but have fancy names so they are more likely to be tested. Just know that the omphalomesenteric duct cyst is usually peripheral, and the allantoic cyst is usually central. If the cysts persist into the 2nd or 3rd trimester then they might be associated with trisomy 18 and 13. You should look close for other problems.

Congenital Fetal Anomalies

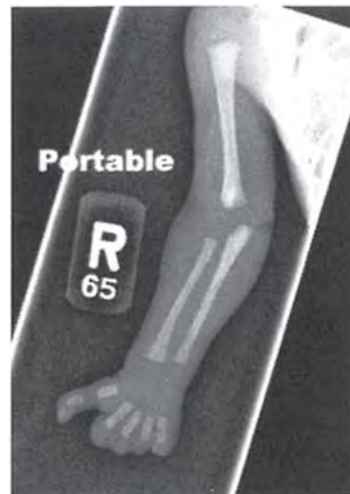
Downs:

Ultrasound Findings Concerning for Downs Syndrome	
Congenital Heart Disease	More than half of fetuses (or fet, if you prefer) with Downs have congenital heart issues, - most commonly AV canal and VSD
Duodenal Atresia	Most common intra abdominal pathology associated with Downs (hard to see before 22 weeks)
Short Femur Length	Not Specific
Echogenic Bowel	Not Specific (can be seen with obstruction, infection, CF, ischemia, and lots of other stuff)
Choroid Plexus Cyst	Not Specific, and actually seen more with Trisomy 18. It should prompt a close survey for other findings (normal if in isolation)
Nuchal Translucency	Translucency > 3mm in the first trimester,
Nuchal Fold Thickness	Thickness > 6mm in the second trimester- nonspecific and can also be seen with Turners
Echogenic Focus in Cardiac Ventricle	Not Specific, but increased risk of Downs x 4

Nuchal Lucency: Measured between 9-12 weeks, this anechoic area between the neck/ occiput and the skin **should be less than 3mm**. Measurements > 3mm are associated with Downs (trisomy 21) or other chromosomal abnormalities. Positioning of the neck is critical to avoid false positives.

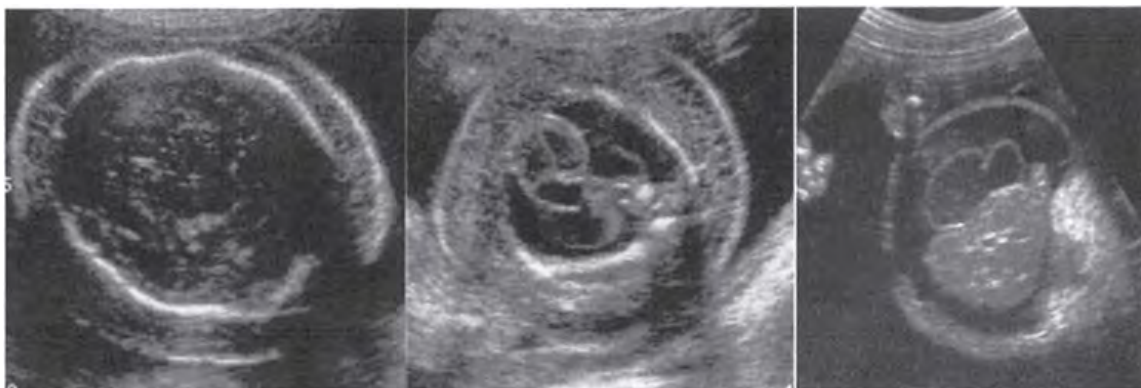


Amniotic Band Syndrome: The fetus needs to stay in the amniotic cavity, and stay the hell out of the chorionic cavity. If the amnion gets disrupted and the fetus wonders / floats into the chorionic cavity he/she can get caught in the sticky fibrous septa. All kinds of terrible can result ranging from decapitation, to arm/leg amputation. This is most likely to be shown in one of two ways: (1) a x-ray of a hand or baby gram showing fingers amputated or a hand/arm amputated - with the remaining exam normal, or (2) a fetal ultrasound with the bands entangling the arms or legs of a fetus.



Amniotic Bands Syndrome
-Amputated Fingers

Hydrops: Fetal hydrops is bad news. This can be from immune or non-immune causes. The most common cause is probably Rh sensitization from prior pregnancy. Some other causes include; TORCHS, Turners, Twin Related Stuff, and Alpha Thalassemia. Ultrasound diagnosis is made by the presence of two of the following: **pleural effusion, pericardial effusion,** and Subcutaneous Edema. A sneaky trick is to instead show you a thickened placenta (> 4-5cm), although I think it's much more likely to show a pleural effusion and pericardial effusion.



Hydrops – Body Wall Edema, Pleural Effusion, Ascites

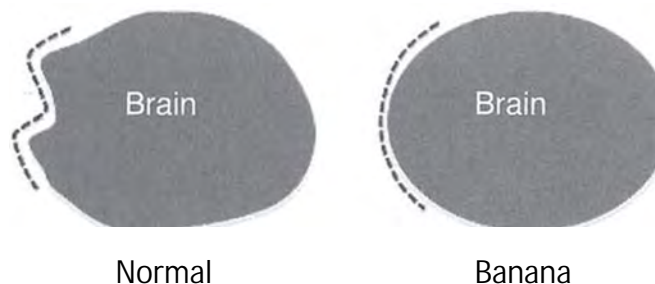
Lemon Sign - The appearance of an indented frontal bone, classically seen as a sign of Chiari II, although also seen a lot in spina bifida (like 90% of the time). You typically see this before 24 weeks (it often **disappears after 24 weeks**).



Lemon Sign - Indented Frontal Bone

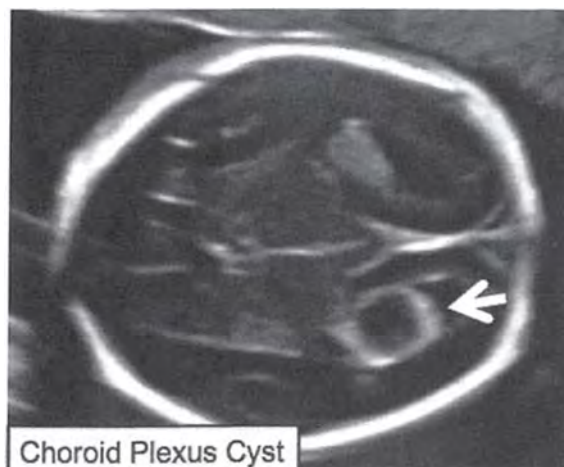
Banana Sign - The way the cerebellum wraps around the brainstem as the result of spinal cord tethering (and downward migration of the posterior fossa) looks like a banana. In other words, the cisterna magna is obliterated and the cerebellum looks like a banana. Just like the lemon sign this is seen with **Chiari II and Spinal Bifida**.

Anterior curving of the cerebellar hemispheres with simultaneous obliteration of the cisterna magna is the so called “banana sign.” Supposedly, the idea is that you have a neural tube defect, which lets you leak CSF from the spinal defect. Once you leak enough, you get hypotension in the subarachnoid space, with prolapse of the cerebellum into the foramen magnum.



Other findings of spina bifida include a small biparietal diameter and ventricular enlargement.

Choroid Plexus Cyst - This is one of those incidental findings that in isolation means nothing. Having said that, the incidence of this finding is increased in trisomy 18, trisomy 21, Turner’s Syndrome, and Klinefelter’s.



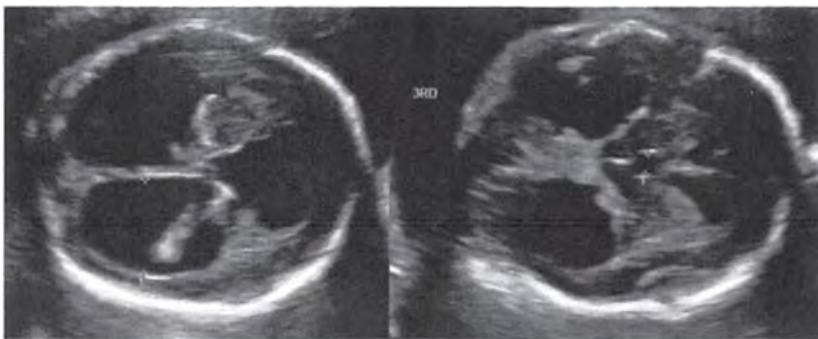
Facial Clefts - This is the most common fetal facial anomaly. About 30% of the time you are dealing with a chromosome anomalies. Around 80% of babies is cleft lips have cerebral palsy. You can see cleft lips, but cleft palate (in isolation) is very hard to see.

Cystic Hygroma - If they show you a complex cystic mass in the posterior neck, in the antenatal period, this is the answer. The follow up is the association with Turners and Downs.

Ventriculomegaly - There are multiple causes including; hydrocephalus (both communicating and non-communicating), and cerebral atrophy. Obviously this is bad, and frequently associated with anomalies.

Things to know:

- Aqueductal Stenosis is the most common cause of non-communicating hydrocephalus in a neonate
- Ventricular atrium diameter $> 10\text{mm}$ = too big
- “Dangling choroid” hanging off the wall more than 3mm = too big



Ventriculomegaly- shows dangling choroid
-this was caused by aqueductal stenosis

Anencephaly - This is the most common neural tube defect. You have total **absence of the cranial vault and brain above the level of the orbits**. Obviously this is not compatible with life.



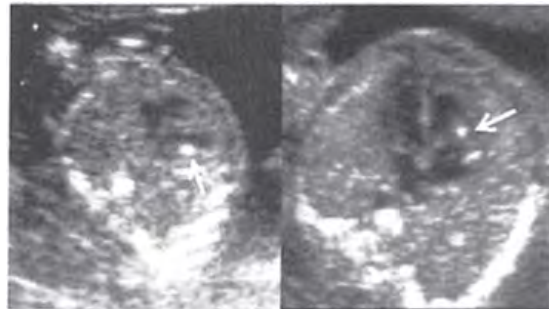
Pleural Effusion - There are multiple causes of pleural effusion in the neonate, but when you see it **I** want you to think **Hydrops**. *The way they show hydrops in case conference / case books and very likely multiple choice is pleural effusions, pericardial effusions, and ascites.*

Congenital Diaphragmatic Hernia - Abdominal contents pushing into the chest. Nearly all are on the left (85%). The things to know is that it (1) causes a high mortality because of the association with **pulmonary hypoplasia**, and (2) that all the kids are **malrotated** (it messes with normal gut rotation). If they show this it will either be (a) a newborn chest x-ray, or (b) a 3rd trimester MRI.

Echogenic Intracardiac Focus (EIF) - This is a calcification seen in a papillary muscle (usually in the left ventricle). You see them all the time, they don't mean that much but are seen at a higher rate Trisomy 21 (12%) and Trisomy 13. So you are supposed to look for more features.

If they ask you a question about this they will be testing one of two facts :

- (1) *it occurs in the normal general population - around 5%,*
- (2) *it occurs more in Downs patients around -12%.*



Echogenic Intracardiac Focus (EIF)

Abnormal Heart Rate: Tachycardia is defined as a rate > 180 bpm. Bradycardia is defined as a rate < 100 bpm.

Double Bubble: This is described in detail in the Peds Chapter. Just realize this can be shown with antenatal ultrasound, or MRI. It's still duodenal atresia.



Double Bubble - Duodenal Atresia

Echogenic Bowel: This can be a normal variant but can also be associated with significant badness. Normally bowel is isoechoic to the liver. If it's equal to the iliac crest bone then it's too bright. The DDX includes CF, Downs and other Trisomies, Viral Infections, and Bowel Atresia.

Sacroccygeal Teratoma: This is the most common tumor of the fetus or infant. These solid or cystic masses are typically large and found either on prenatal imaging or birth. They can cause mass effect on the GI system, hip dislocation, nerve compression causing incontinence, and high output cardiac failure. Additionally, they may cause issues with premature delivery, dystocia, and hemorrhage of the tumor. They are usually benign (80%). Those presenting in older infants tend to have a higher malignant potential. The location of the mass is either external to the pelvis (47%), internal to the pelvis (9%), or dumbelled both inside and outside (34%).

Autosomal Recessive Polycystic Disease - The classic look is massively enlarged bilateral kidneys with oligohydramnios. Additional details in the Peds chapter.

Posterior Urethral Valves: The classic look is bilateral hydro on either fetal US or 3rd Trimester MRI.

Short Femur: A short femur (below the 5th percentile), can make you think of a skeletal Dysplasia.

Maternal Disorders in Pregnancy

Incompetent Cervix: When shortened the cervix is associated with high risk of premature delivery. You call it short when the endocervical canal is $< 2.5\text{cm}$ in length.

Hydronephrosis occurs in 80% of pregnancy (mechanical compression of the ureters is likely the cause). It tends to affect the right more than the left (dextrorotation of the pregnant uterus).

Fibroids: Fibroids tend to grow in the early pregnancy secondary to elevated estrogen. Progesterone will have the opposite effect, inhibiting growth, in later pregnancy. Stretching of the uterus may affect the arterial blood supply and promote infarcts and cystic degeneration.

Things That Grow During Pregnancy:

- *Babies,*
- *Splenic Artery Aneurysms,*
- *Renal AMLs,*
- *Fibroids.*

Uterine Rupture: You see this most commonly in the **3rd trimester at the site of prior c-section**. Other risk factors worth knowing are the unicornuate uterus, prior uterine curettage, “trapped uterus” (persistent retroflexion from adhesions), and interstitial implantation.

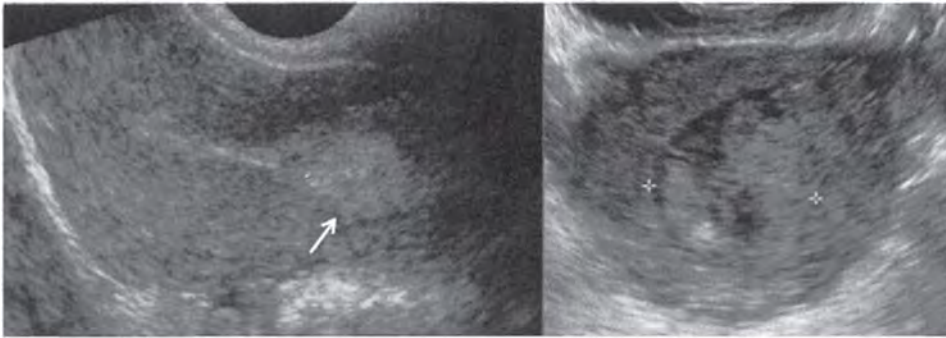
HELLP Syndrome: Hemolysis, Elevated Liver Enzymes, Low Platelets. This is the most severe form of pre-eclampsia, and favors young primigravid women in their 3rd trimester. It's bad news and 20-40% end up with DIC. If they are going to show this, it will be as a subcapsular hepatic hematoma in pregnant (or recently pregnant) women.

Peripartum Cardiomyopathy: This is a dilated cardiomyopathy that is seen in the last month of pregnancy to 5 months post partum. The cardiac MRI findings include a global depressed function, and non-vascular territory subepicardial late Gd enhancement - corresponding to cellular lymphocyte infiltration.

Sheehan Syndrome: This is pituitary apoplexy seen in post-partum female who suffer from large volume hemorrhage. The pituitary grows during pregnancy, and if you have an acute hypotensive episode you can stroke it out. The look on MR is variable depending on the time period, acute it will probably be **T1 bright** (if they show a picture). Ring enhancement around an empty sella is a late look.

Ovarian Vein Thrombophlebitis - This can be a cause of post partum fever. Risk factors include C-section, and endometritis. The right side is affected five times more often than the left. They could show you an enlarged ovary and a thrombosed adjacent ovarian vein.

Retained Products of Conception: The typical clinical story is continued bleeding after delivery (or induced abortion). The most common appearance is an echogenic mass within the uterine cavity. The presence or absence of flow is variable, you can have lots or you can have none. A sneaky way to show this is irregular thickening of the endometrium (>1 Omm) with some reflective structures and shadowing - representing the fetal parts. You can also think about RPOC when the endometrial thickness is > 5 mm following dilation and curettage. Testable associations include: medical termination of pregnancy (abortion), second trimester miscarriage, and placenta accreta.

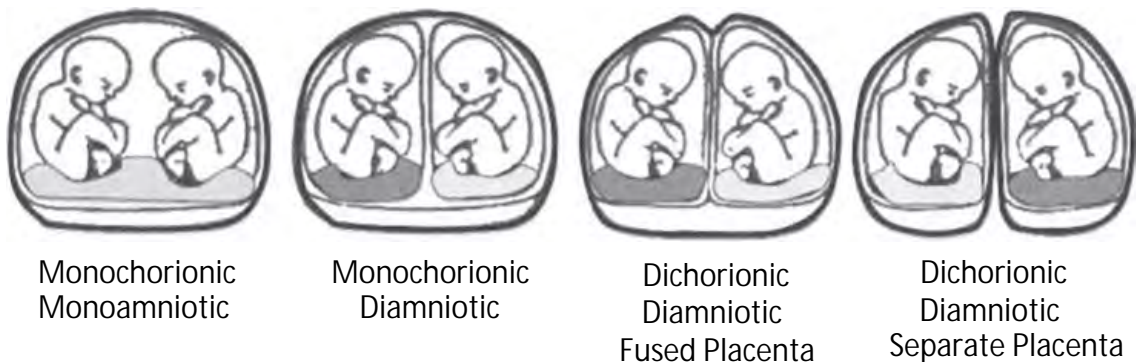


Retained Products of Conception

Endometritis - Broadly speaking it is an inflammation or infection of the endometrium. The history will be (if you are given one) fever and uterine tenderness and c-section (or prolonged labor). On ultrasound you will see a thickened, heterogenous endometrium, with or without fluid / air.

Multiple Gestations

Placentation Terminology: So you can have monozygotic twins (identical), or dizygotic twins (not-identical). The dizygotics are always dichorionic and diamniotic. The placenta of the monozygotics is more variable and depends of the timing of fertilized ovum splitting (before 8 days = diamniotic, after 8 days = monoamniotic). As a point of trivia, a late splitting (after 13 days) can cause a conjoined twin. As a general rule the later the split the worse things do (monoamniotics have more bad outcomes - they get all tangled up, and conjoineds have even more problems).

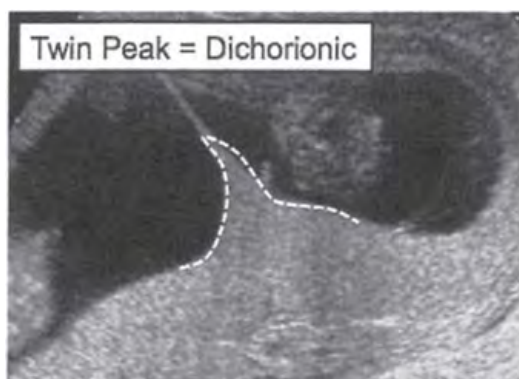


Monochorionic vs Dichorionic

Membrane Thickness: To differentiate the different types, some people use a method classifying thin and thick membranes. Thick = “easy to see” 1-2 mm, Thin = “hard to see.” Thick is supposed to mean there are 4 layers (dichorionic). Thin is supposed to be 2 layers (monochorionic). Obviously this method is very subjective

Twin-Peak Sign: A beak-like tongue between the two membranes of a dichorionic diamniotic fetuses. This **excludes a monochorionic pregnancy.**

T Sign: This is essentially the absence of the twin peak sign. You don’t see chorion between membrane layers. **T sign = monochorionic pregnancy.**



Twin Growth - You can use normal growth charts in the first and second trimester (but not the third). The femur length tends to work best for twin age in the later pregnancy. More than 15% difference in fetal weight or abdominal circumference between twins is considered significant.

Twin- Twin Transfusion - This occurs in monochorionic twins when a vascular communication exists in the placenta. You end up with one greedy fat twin who takes all the blood and nutrients, and one skinny wimpy looking kid who gets the scraps. The somewhat counter intuitive part is that the skinny kid actually does better, and the fat one usually gets hydrops and dies. You are going to have unequal fluid in the amniotic sacs, with the donor (skinny) twin having severe oligohydramnios and is sometimes (***buzzword**) **“stuck to the w'all of the uterus”**. The fat twin floats freely in his polyhydramniotic sac. The donor (skinny) twin will also have a high resistance umbilical artery spectrum.

Tw'in Reversed Arterial Perfusion Syndrome - You can get intraplacental shunting that results in a “pump twin” who will pump blood to the other twin. The other twin will not develop a heart and is typically referred to as an “acardiac twin.” The acardiac twin will be wrecked (totally deformed upper body). The “pump twin” is usually normal, and does ok as long as the strain on his/her heart isn't too much. If the acardiac twin is really big (> 70% estimated fetal weight of the co-twin) then the strain will usually kill the pump twin. They could show this as a Doppler ultrasound demonstrating umbilical artery flow toward the acardiac twin, or umbilical vein flow away from the acardiac twin.

One Dead Twin - At any point during the pregnancy one of the twins can die. It's a bigger problem (for the surviving twin) if it occurs later in the pregnancy. **“Fetal Papyraceous”** is a fancy sounding Latin word for a pressed flat dead fetus.

“Twin-Twin Embolization Syndrome” is when you have embolized necrotic dead baby being transferred to the living fetus (*soylent green is people!*). This can result in DIC, tissue ischemia, and infarct. By the way, a testable point is that this transfer can only occur in a monochorionic pregnancy.

5

Endocrine

Prometheus Lionhart, M.D.



The endocrine section is a mix of thyroid pathology, adrenal pathology, and a few random syndromes. Remember the list of things that can happen in the adrenals, thyroid, and pancreas is relatively short. The testable trivia is usually straight forward, with the tricky things related to associations.

High Yield Topics

- MEN syndromes - always think about these with a thyroid / pancreas case
- Adrenal Washout Equations
- Pheochromocytoma - MRI characteristics, syndromic associations

Adrenal

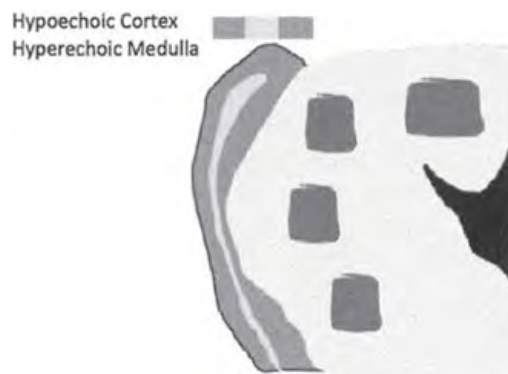
Anatomy: The adrenal glands are paired retroperitoneal glands that sit on each kidney. The right gland is triangular in shape, and the left gland tends to be more crescent shaped. If the kidney is congenitally absent the glands will be more flat, straight, discoid, or “*pancake*” in appearance. Each gland gets arterial blood from three arteries (superior from the inferior phrenic, middle from the aorta, and inferior from the renal artery). The venous drainage is via just one main vein (on the right into the IVC, on the left into the left renal vein).



Step 1 Trivia: There are 4 zones to the adrenal, each of which makes different stuff.

- * *Zona Glomerulosa*: Makes Aldosterone - prolonged stimulation here leads to hypertrophy.
- * *Zona Fasciculata*: More Cortisol
- * *Zona Reticularis* - Makes Androgens
- * *Medulla* - Makes Catecholamines

Normal Look on Ultrasound: In babies you can actually see the adrenal on ultrasound. If they show you a PEDS adrenal case it's gonna be on ultrasound (or MIBG). If it's an adult case it will be CT or MRI. In babies the cortex is hypoechoic, and the medulla is hyperechoic. This gives the adrenal a **triple stripe appearance (dark cortex, bright medulla, dark cortex)**.



Hypertrophy:

- * **21-Hydroxylase Deficiency:** Congenital adrenal hypertrophy is caused by **21-hydroxylase deficiency** in > 90% of cases. It will manifest clinically as either **genital ambiguity (girls)** or some salt losing pathology (boys). The salt losing can actually be life threatening. The look on imaging is adrenal limb width greater than 4mm, and loss of the central hyperechoic stripe.
- * **Cushing Syndrome:** Too much cortisol. This is most commonly the result of a pituitary adenoma (75%), or ectopic production from a small cell lung cancer. In these cases you are going to see **bilateral adrenal gland hyperplasia**. *Less commonly (20%) it is from an adrenal adenoma.*

Hemorrhage: This occurs most commonly in the setting of trauma, or stress (neonates).

- * **Stress:** It's classically seen after a breech birth, but can also be seen with fetal distress, and congenital syphilis. Imaging features change based on the timing of hemorrhage. It will evolve from hyperechoic to isoechoic to hypoechoic. Calcification is often the end result. It should be avascular. This can occur bilaterally, but favors the right side (75%). Serial ultrasounds (or MRI) can differentiate it from a cystic neuroblastoma. The hemorrhage will get smaller (the cancer will not).
- * **Trauma:** This is going to be an adult (in the setting of trauma). Most likely it will be shown on CT. It's more common on the right.

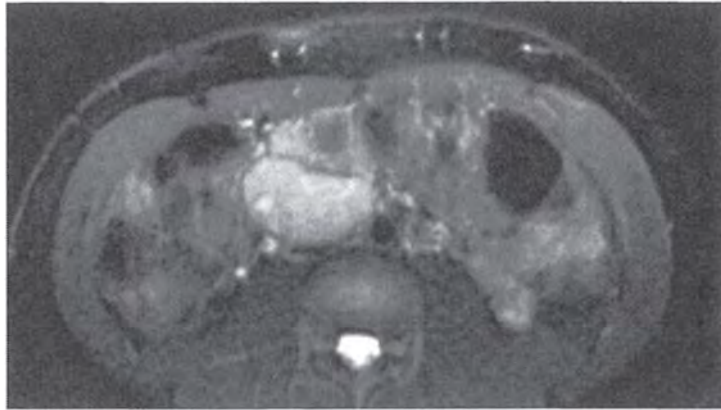


* **Zebra: Waterhouse-Friderichsen Syndrome** - Hemorrhage of the adrenal in the setting of fulminant meningitis (from *Neisseria Meningitidis*).

Masses (other than adenoma).

* **Pheochromocytoma** - Uncommon in real life (common on multiple choice tests).

They are usually large at presentation (larger than 3cm). It's usually a heterogenous mass on CT. On MRI they are **T2 bright**. Both MIBG and Octreotide could be used (but MIBG is better since Octreotide also uptakes in the kidney).



Pheo at *Organ of Zuckerkandl* - T2 Bright

O Trivia: "Rule of 10s"

10% are extra adrenal (organ of Zuckerdandl - usually at the IMA),
10% are bilateral, 10% are in children, 10% are hereditary, 10% are
NOT active (no HTN).

o Trivia: "Syndromes"

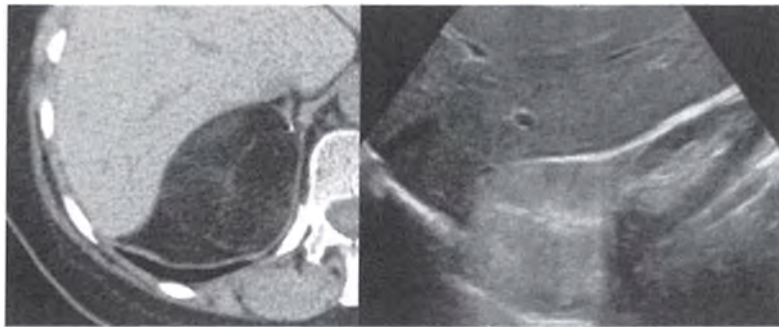
Associated syndromes: First think **Von Hippel Lindau**, then think
MEN IIa and lib. Other things less likely to be tested include NF-1,
Sturge Weber, and TS.



o Trivia: "Carney Triad"

Extra-Adrenal Pheo, GIST, and Pulmonary Chondroma (hamartoma).
*Don't confuse this with the Carney Complex (Cardiac Myxoma, and
Skin Pigmentation).*

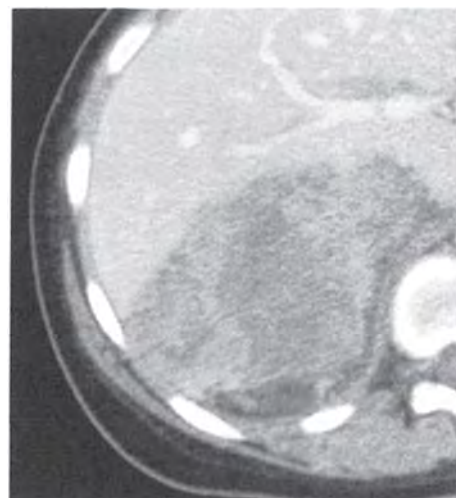
- **Neuroblastoma** - *Discussed extensively in the Peds chapter.*
- * **Myelolipoma** - Benign tumor that **contains bulk fat**. About 1/4 have calcifications. If they are big (> 4cm) they can bleed, and present with a retroperitoneal hemorrhage. Another piece of trivia is the association with endocrine disorders (Cushings, Congenital Adrenal Hyperplasia, Conns). Don't get it twisted, these tumors are NOT functional, they just happen to have associated disorders about 5-10% of the time.



Myelolipoma - Contains bulk fat, Hyperechoic on US

- * **Cyst** - You can get cysts in your adrenal. They are often unilateral, and can be any size. The really big ones can bleed. They have a thin wall, and do NOT enhance.
- * **Mets:** Think **breast, lung, and melanoma**. They have no specific imaging findings and look like lipid poor adenomas. If the dude has a known primary (especially lung, breast, or melanoma), and it's not an adenoma then it's probably a met. *

- * **Cortical Carcinoma:** These are **large** (4cm-10cm), maybe functional (Cushings), and **calcify in about 20% of cases**. They are bad news and often met everywhere (direct invasion often first). As a pearl, an adrenal carcinoma is not likely to be less than 5cm and often has central necrosis.



Adrenal Cortical Carcinoma

- *Direct Invasion of the liver*

Adenoma:

These things are super common, and are easily the most common tumor in the adrenal gland. Up to 8% of people have them. Proving it is an adenoma is an annoying problem.

- * Non-Contrast: Less than 10 HU
- * Contrast: Two options:

Absolute:

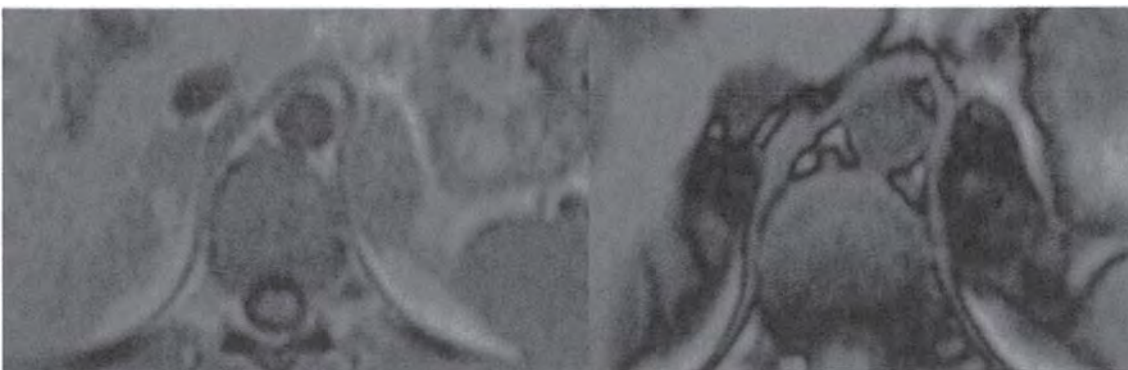
$$\frac{\text{Enhanced CT} - \text{Delayed CT}}{\text{Enhanced CT} - \text{Unenhanced CT}} \times 100 \quad \text{Greater than 60\% = Adenoma}$$

Relative:

$$\frac{\text{Enhanced CT} - \text{Delayed CT}}{\text{Enhanced CT}} \times 100 \quad \text{Greater than 40\% = Adenoma}$$

- * MRI: Look for drop out on in and out of phase T1.

Trivia: Although most adenomas are not functional, Cushings (too much cortisol) and Conn (too much aldosterone) can present as a functional adenoma.



Adrenal Adenoma - Signal drop out In and Out of Phase

Conn's Syndrome: Syndrome of excessive aldosterone production. This is **most commonly caused by a benign adenoma** (70%). Cortical-carcinoma can also do it, but that is much more rare and usually accompanied by hypercortisolism.

Calcifications:

This is often the result of prior trauma or infection (TB). Certain tumors (cortical carcinoma, neuroblastoma) can have calcifications. Melanoma mets are known to calcify.



Wolman Disease: - This is a **total Aunt Minnie** (and massive zebra).

Bilateral enlarged calcified adrenals. It's a fat metabolism error thing that kills within 6 months. The disease usually kills before the first year of life.

Syndromes:

MEN: "Multiple Endocrine Neoplasia"

There are three of these stupid things, and people who write multiple choice tests love to ask questions about them.

- * **MEN 1:** Parathyroid Hyperplasia (90%), Pituitary Adenoma, Pancreatic Tumor (Gastrinoma most commonly)
- * **MEN 2:** Medullary Thyroid Cancer (100%), Parathyroid hyperplasia, Pheochromocytoma (33%) *
- * **MEN 2b:** Medullary Thyroid Cancer (**80%**), Pheochromocytoma (**50%**), Mucosal Neuroma, Marfanoid Body Habitus

MEN Mnemonic

MEN I (3Ps)

-Pituitary, Parathyroid, Pancreas

MEN IIa (1M, 2Ps)

Medullary Thyroid Ca,
Pheochromocytoma, Parathyroid

MEN IIb (2Ms, 1P)

- Medullary Thyroid Ca, Marfanoid Habitus /mucosal neuroma,
Pheochromocytoma

Carcinoid Syndrome: Flushing, diarrhea, pain, right heart failure from serotonin manufactured by the carcinoid tumor. The syndrome **does not occur until the lesion mets to the liver** (normally the liver metabolizes the serotonin). The typical primary location for the carcinoid tumor is the GI tract (70%), with the appendix being the overall most common location. The actual syndrome only occurs in 10% of cases - and is actually very rare (in real life - not on tests). Another piece of trivia worth knowing is the association of GI carcinoids with other GI tumors (GI adenocarcinoma).



Carcinoid - Classic Mesenteric Involvement

Thyroid:

Anatomy: The thyroid gland is a butterfly shaped gland, with two lobes connected by an isthmus. The thyroid descends from the foramen cecum at the anterior midline base of the tongue along the thyroglossal duct. The posterior nodular extension of the thyroid (Zuckerkandl tubercle) helps give a location of the recurrent laryngeal nerve (which is medial to it).

Thyroid Nodules: Usually evaluated with ultrasound. Nodules are super super common and almost never cancer. This doesn't stop Radiologists from imaging them, and sticking needles into them. Ultrasound guided FNA of colloid nodules is a major cash cow for many body divisions, that on very rare occasions will actually find a cancer. Qualities that make them more suspicious include: more solid (cystic more benign), calcifications (especially microcalcifications). **Microcalcifications are supposed to be the buzzword for papillary thyroid cancer.** "Comet Tail" artifact is seen in Colloid Nodules. "Cold Nodules" on 1-123 scans are still usually benign but have cancer about 15% of the time, so they actually deserve workup.

Thyroglossal Duct Cyst - This can occur anywhere between the foramen cecum (the base of the tongue) and the thyroid gland. They are usually found in the **midline at or above the hyoid**. It looks like a thin walled cyst.

Why care?

- * They can get infected
- Rarely they can have papillary thyroid cancer (if you see an enhancing nodule)



Thyroglossal Duct Cyst - Midline

Ectopic and Lingual Thyroid Similar to a thyroglossal duct cyst, this can be found anywhere from the base of the tongue through the central neck. The **most common location (90%) is the tongue base ("Lingual Thyroid")**. It will look hyperdense because of its iodine content (just like a normally located thyroid gland). If you find this, make sure you check for a normal thyroid (sometimes this is the only thyroid the dude has). As a point of trivia, the rate of malignant transformation is rare (3%).

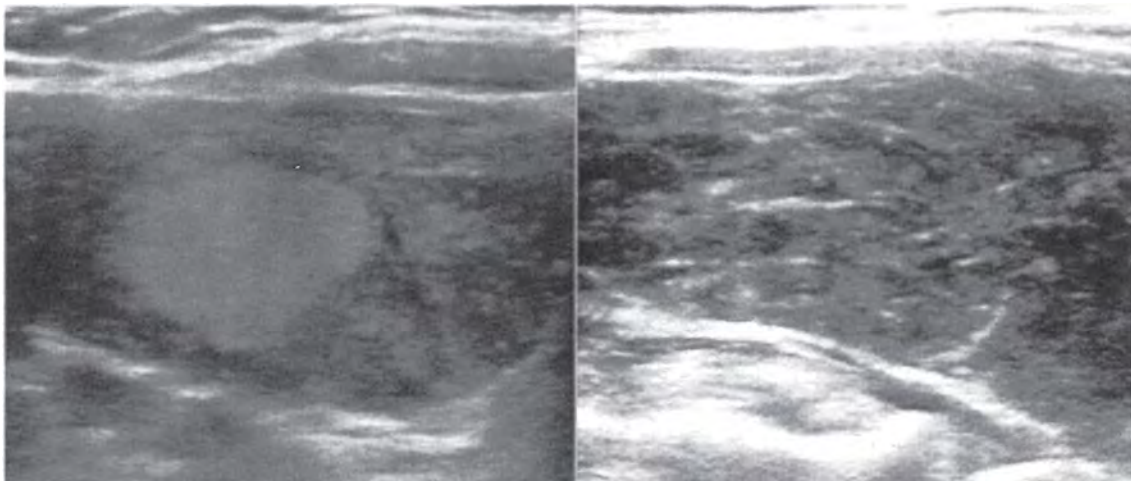
Goiter - Thyroid that is too big. In North America it's gonna be a multi-nodular goiter or graves. In Africa it's low iodine. You can get compressive symptoms if it mashes the esophagus or trachea.

Graves - Autoimmune disease that causes hyperthyroidism (most common cause). It's primary from an antibody directed at the TSH receptor. The actual TSH level will be low. The gland will be enlarged and "inferno hot" on Doppler.

- *Graves Orbitopathy*: Spares the tendon insertions, doesn't hurt (unlike pseudotumor). Also has increased intra-orbital fat.
- * *Nuclear Medicine*: Increased uptake of 1-123 %RAIU usually 50-80%. Visualization of pyramidal lobe is accentuated.

Hashimotos - The **most common cause of goitrous hypothyroidism** (in the US). It is an autoimmune disease that causes hyper then hypo thyroidism (as the gland bums out later). It's usually hypo - when it's seen. It has an **increased risk of primary' thyroid lymphoma**. Step 1 trivia; associated with autoantibodies to thyroid peroxidase (TPO) and antithyroidglobulin..

On Ultrasound: There are two classic findings (a) the heterogeneous "giraffe skin" appearance, (b) white knights - uniform hyperechoic nodules - which are actually regenerative nodules.



White Knight

Giraffe Skin

Subacute Thyroiditis /De Quervains Thyroiditis: The classic clinical scenario is a female with a painful gland after an upper respiratory infection. There is a similar subtype that happens in pregnant women, although this is typically painless. You get hyperthyroidism (from spilling the hormone) and then later hypothyroidism. As you get over your cold, the gland recovers to normal function. Radiotracer uptake will be decreased during the acute phase.

Reidels Thyroiditis - This is one of those IgG4 associated diseases (others include orbital pseudotumor, retroperitoneal fibrosis, sclerosing cholangitis). You see it in women in their 40s-70s. The thyroid is replaced by fibrous tissue and diffusely enlarges causing compression of adjacent structures (dysphagia, stridor, vocal cord palsy). On US there will be decreased vascularity. On an uptake scan you are going to have decreased values. A sneak trick would be to show you a MR (it's gonna be dark on all sequences - like a fibroma).

Acute Suppurative Thyroiditis: This is an actual bacterial infection of the thyroid. It is possible to develop a thyroid abscess in this situation. A unique scenario (highly testable) is that **in kids this infection may start in a 4th brachial cleft anomaly** (usually on the left), travel via a pyriform fistula and then infect the thyroid. Honestly, that is probably too much for the CORE - but could show up on a certification exam under neuro.

Colloid Nodules: These are super super common. Suspicious features include microcalcifications, and increased vascularity, solid size (larger than 1.5cm), and being cold on a nuclear uptake exam. **Comet artifact is the buzzword.**

Thyroid Adenoma: These look just like solid colloid nodules on ultrasound. They can be hyper functioning (hot on uptake scan). Usually if you have a hyper-functioning nodule, your background thyroid will be colder than normal (which makes sense).

Thyroid Cancer: You can get lots of cancers in your thyroid. There are 4 main subtypes of primary thyroid cancer. Additionally you can get mets to the thyroid or lymphoma in your thyroid - this is super rare and I'm not going to talk about it.

Papillary	The Most Common Subtype. " <i>Papillary is Popular</i> "	Microcalcifications is the buzzword and key finding (seen in the cancer and nodes).	Mets via the lymphatics. Has an overall excellent prognosis, and responds well to 1-131.
Follicular	The second most common subtype.		Mets hematogenously to bones, lung, liver, etc.. Survival is still ok, (less good than papillary). Does respond to 1-131.
Medullary	Uncommon	Association with MEN II syndrome. <i>Calcitonin production is a buzzword.</i>	Tendency towards local invasion, lymph nodes, and hematogenous spread. Does NOT respond to 1-131.
Anaplastic	Uncommon	Seen in Elderly. Seen in people who have had radiation treatment.	Rapid growth, with primary lymphatic spread. Does NOT respond to 1-131.
Hurthle Cell (variant of Follicular)	Uncommon	Seen more in Elderly.	Does not take up 1-131 as well as normal follicular. <i>FDG-PET is the way to go for surveillance.</i>

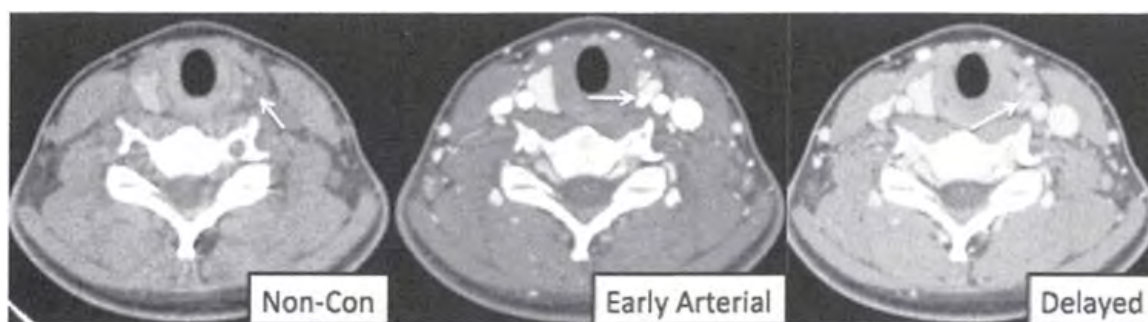
Metastasis: The buzzword is going to be **microcalcifications in a node** (with papillary).

The nodes are typically hyperechoic compared to regular nodes, hyperenhancing on CT, and T1 bright on MR. Remember that thyroid cancer is hyper vascular, and it can bleed like stink when it mets to the brain. If there are **mets to the lungs the classic pattern is “miliary.”** The additional pearl with regard to lung mets is that they can be occult on cross sectional imaging, and only seen on whole body scintigraphy. For the purpose of multiple choice tests *pulmonary fibrosis is a risk of treating with 1-131 if you have diffuse lung mets.*

Parathyroid

Anatomy: There are normally 4 parathyroid glands located posterior to the thyroid. The step 1 trivia is that the superior 2 are from the 4th branchial pouch, and the inferior 2 are from the 3rd branchial pouch. The inferior two are more likely to be in an ectopic location.

Parathyroid Adenoma - This is by far the **most common cause of hyperparathyroidism** (90%). On ultrasound these things look like hypoechoic beans posterior to the thyroid. A 4D-CT can be used to demonstrate early wash in and delayed wash out. Nuclear medicine can use two techniques (1) the single tracer, dual phase Sestamibi, or (2) the dual tracer Sestamibi + 1-123 (or Pertechnetate). These are discussed in detail in the nukes section.



Parathyroid Adenoma - 4D CT shows early enhancement and washout

Parathyroid Carcinoma - This is pretty uncommon, and only makes up about 1% of the causes of hyperparathyroidism. It looks exactly like an adenoma on imaging. The only way you can tell on imaging is if they show you cervical adenopathy or invasion of adjacent structures.

High Yield Parathyroid Trivia:

Q: What are the causes of hyperparathyroidism?

A: *Hyperfunctioning Adenoma* (85-90%), Multi-Gland Hyperplasia (8-10%), Cancer (1-3%).

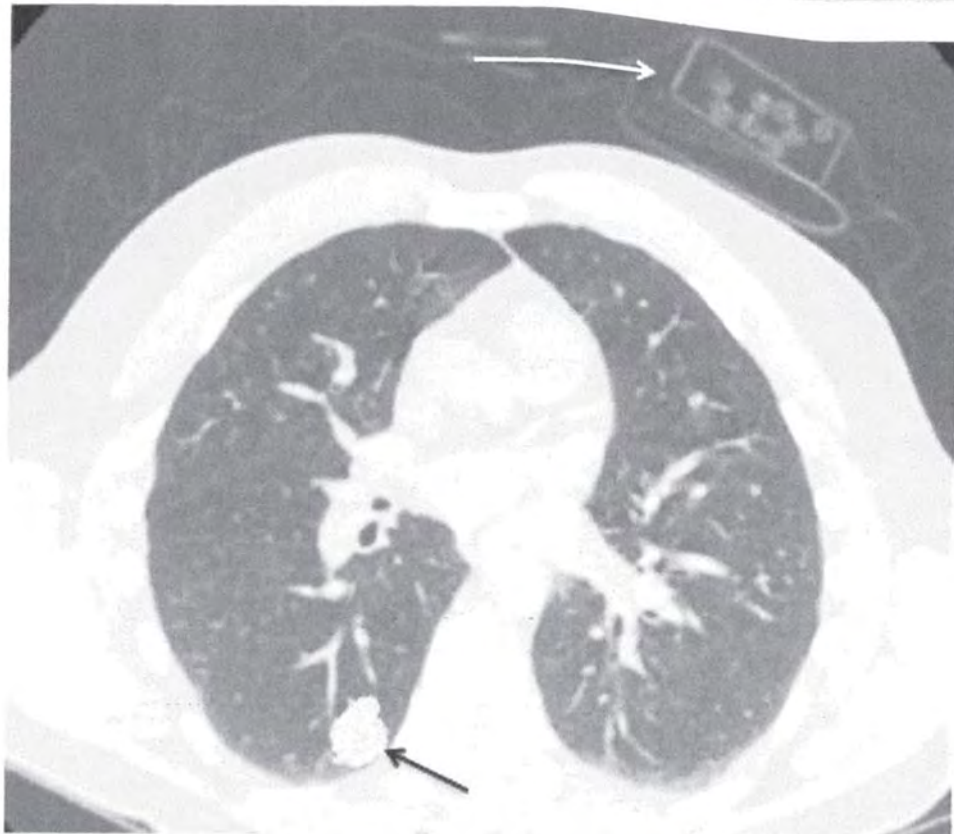
Q: What factors does sestamibi parathyroid imaging depend on ?

A: Mitochondrial density, and blood flow

6

Thoracic

Prometheus Lionhart, M.D.



Because so many things in the chest look exactly the same and differential questions just do not work well on multiple choice, question writers are left with basically two options. The first is to show you an Aunt Minnie. The second is to either tell you or make it obvious what something is, then ask a trivial associated fact.

High Yield Topics:

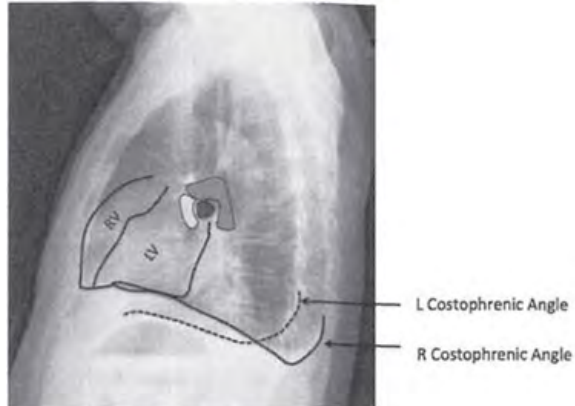
- Atelectasis Patterns
- TB
- Anything HIV / AIDS Related
- Interstitial Lung Disease - Especially associations
- Upper vs Lower Lobe Predominance

Anatomy

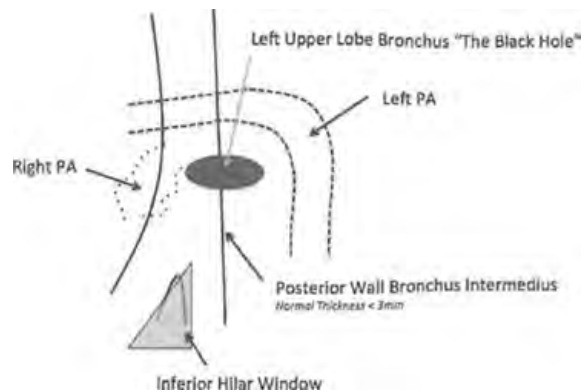
Anatomy is always high yield.

The Lateral CXR “The Radiologists View”

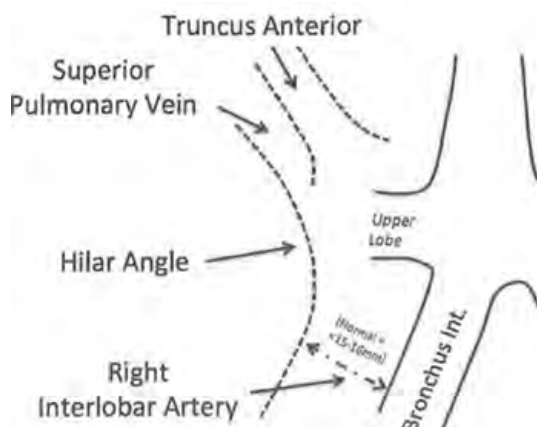
Right Ribs vs Left Ribs on Lateral CXR: By convention, lateral CXRs are taken in the left lateral position (left side against the x-ray film/cassette). Therefore, the left ribs will not be magnified (**rib ribs will be magnified**). **Right ribs also project more posteriorly**. Another strategy is to follow the diaphragm over the stomach bubble (usually left sided).



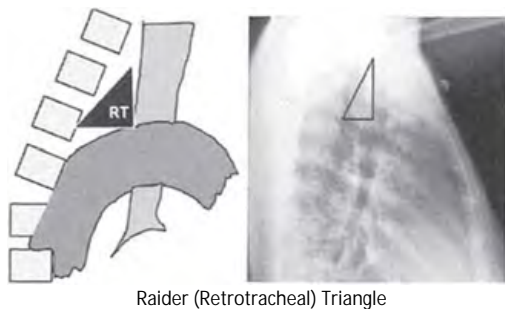
Normal Hilum on Lateral: If you put your finger in the “Dark Hole” - which is the left upper lobe bronchus, in front of it will be the right PA, and overtop of it will be the left PA. The posterior wall of the bronchus intermedium runs through the black hole, and can be thickened by edema.



This is the right hilar anatomy on the frontal view. Of course it never looks that nice in the real world. Ben Felson used to say the right interlobar artery reminded him of a woman's leg... but then again most things did.

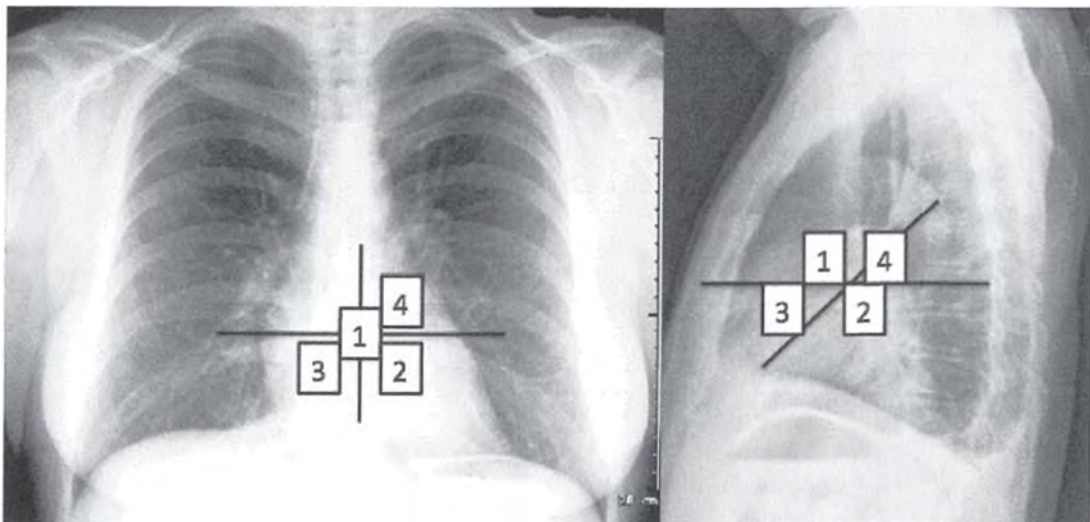


Retrotracheal Triangle (Raider Triangle).



This is a triangle which sits on the aortic arch and is bordered anteriorly by the back wall of the trachea, and posteriorly by the upper thoracic vertebral bodies. Many things can obliterate this, but for the purpose of multiple choice tests an opacity in the Raider Triangle is an **Aberrant right subclavian artery**.

Heart Valves on CXR: This is high yield. I like to use a two intersecting line method on both the frontal and lateral chest to answer this kinds of questions.

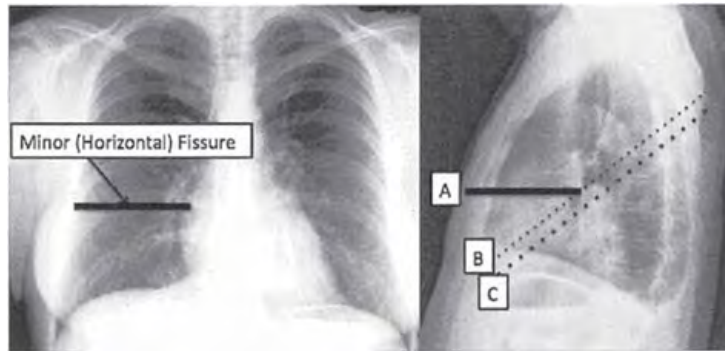


1) Aortic Valve, 2) Mitral Valve, 3) Tricuspid Valve 4) Pulmonic Valve

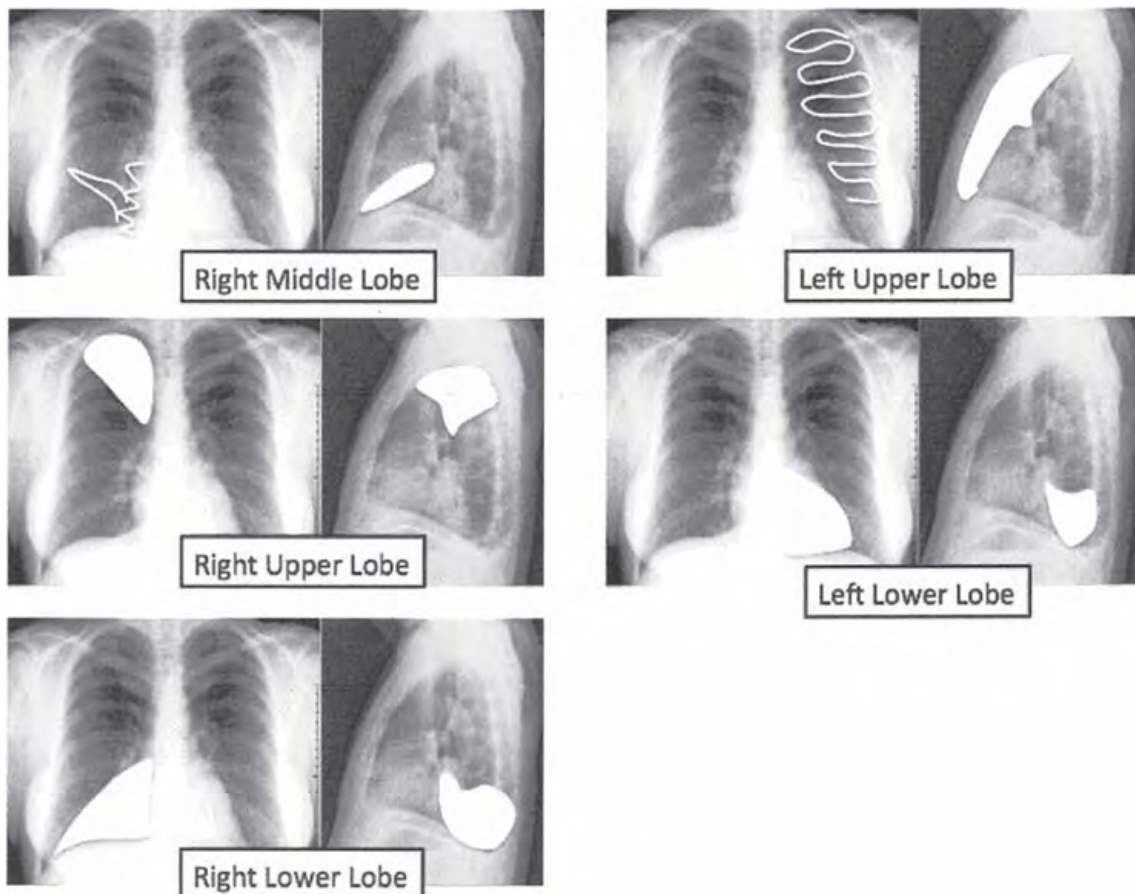
A few other sneaky tricks include, knowing that the pointy parts of the mechanical valves (*Carpentier-Edwards aortic valve*) point out (towards the direction of blood flow). Know that the mitral valve is larger than the aortic valve (so if you see two metallic rings, the larger is the mitral). Know that a pacemaker wire going through a valve makes it the tricuspid valve (lead terminates in the right ventricle). Know the Pulmonic valve is the most superior in location.

Fissures and Atelectasis:

Notice the right major fissure is anterior to the left.



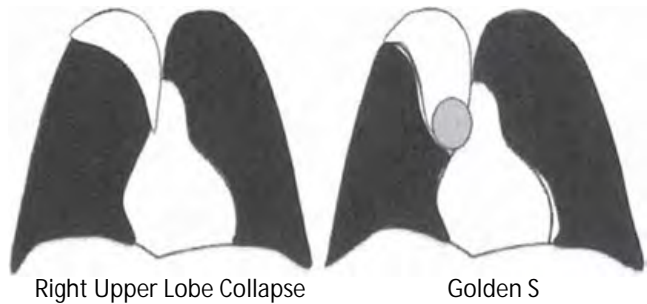
A= Horizontal (Minor) Fissure, B = Right Major, C = Left Major



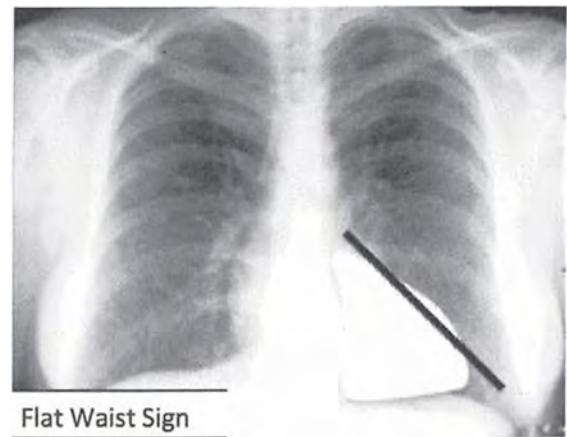
Above are the classic patterns of atelectasis.

A few pearls to point out:

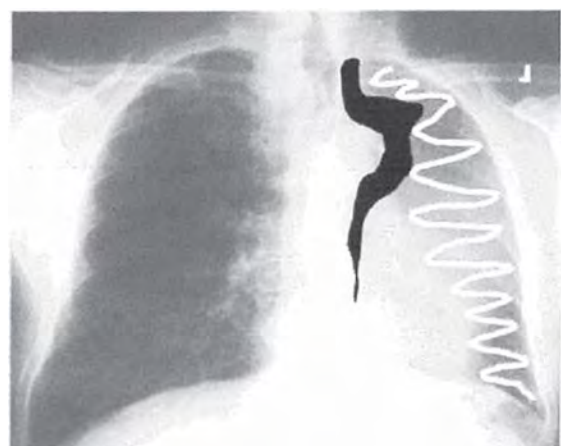
*Right Upper Lobe Collapse = Be aware of the "**Golden S**" type of collapse which infers a central mass.*



The “Flat Waist Sign ” of Left Lower Lobe Collapse. For some reason academic Radiologists are obsessed with the name of this sign.

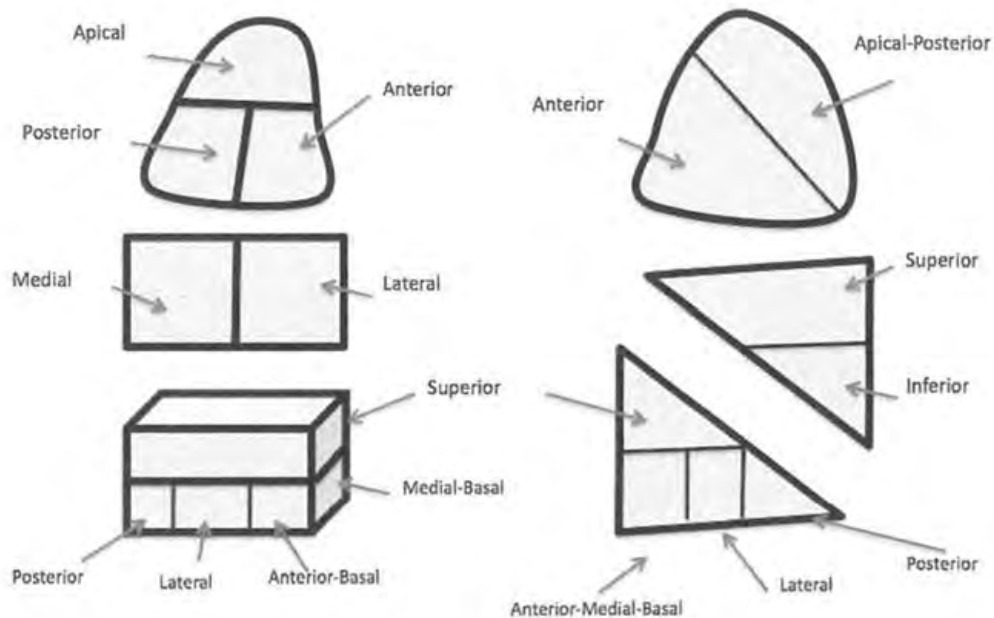


Luftsichel Sign: The is seen in the case of left upper lobe collapse, where you get hyperinflation of the superior segment of the left lower lobe. This inflated segment takes the shape of a sickle (made of air). Luft = Air.



Luftsichel Sign

Segmental Anatomy - The tertiary bronchi are grouped into bronchopulmonary segments. There are 10 segments on the right (3 upper, 2 middle, and 5 lower). On the left there are only 8 segments (4 in the upper lobe / lingula, and 4 in the lower lobe).



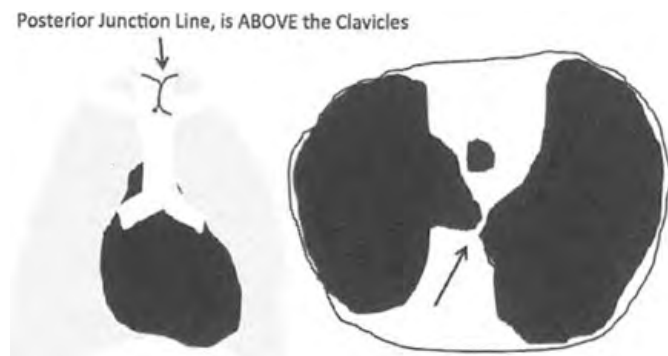
Mediastinum

Anatomy: The mediastinum is classically divided into 4 sections, superior, anterior, middle, and posterior. The borders of these areas make good trivia questions.

Borders:

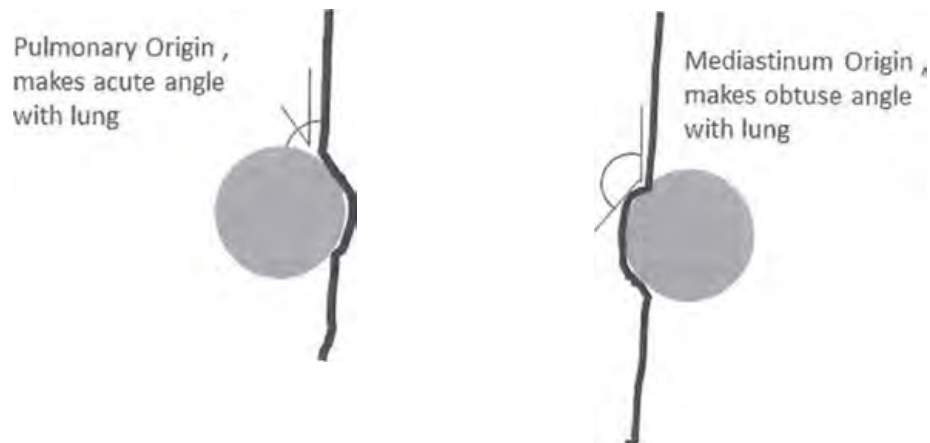
- * *Superior* - The inferior border is the oblique plane from the sternal-manubrial junction.
- * *Anterior* - The posterior border is the pericardium
- * *Middle* - The heart, pericardium, and bifurcation of the trachea are all included. On lateral CXR, people sometimes say posterior to the trachea, and anterior to the vertebral bodies (or 1cm posterior to the vertebral bodies). *
- * *Posterior* - From the back of the heart to the spine. Contains the esophagus, thoracic duct, and descending aorta.

Cervicothoracic Sign - This takes advantage of the posterior junction line, which demonstrates that *things above the clavicles are in the posterior mediastinum*.



Hilum Overlay Sign: Mass at the level of the hilum arising from the hilum will obliterate the silhouette of the pulmonary vessels. If you can see the edge of the vessels through the mass, then the mass is not in the hilum (so it is either anterior or posterior).

Origin: Before you start, you have to make sure it is a mediastinal lesion (if you only have plain films). The mass will make an acute angle with the lung if it's within the lung. The mass will make an obtuse angle with the lung if it's in the mediastinum.



Varient Anatomy:

Azygos Lobe - These things happen when the azygos vein is displaced laterally during development. The result is a deep fissure in the right upper lobe. It's not actually an accessory lobe but rather a variant of the right upper lobe. If they show you one, the question is most likely going to be, "*how many layers of pleura?* " The answer is 4.

Pig Bronchus/ Tracheal Bronchus: A tracheal bronchus is a bronchus that comes right off the trachea (prior to bifurcation into right and left mainstem). You can call it a pig bronchus if the entire right upper lobe is supplied by this bronchus. It usually means nothing clinically, but occasionally people can get some air trapping or recurrent infections from impaired ventilation.

Pulmonary Veins: Pulmonary vein anatomy is highly variable. You typically have 4 total (2 right - upper and lower, 2 left - upper and lower). The **most common anatomic variation is a separate vein draining the right middle lobe** (seen 30% of the time). Who cares??? Two people (1) People who write multiple choice tests, (2) Electrophysiologists prior to ablations.

Proximal Interruption of the Pulmonary Artery: Basically you have congenital absence of the right (or left PA) with the more distal pulmonary vasculature present. It's also called unilateral absence of the PA, but that is confusing because the distal pulmonary vasculature is present.

How it's shown:

- Classically with volume loss of one hemi-thorax (could be on CXR or CT), then a contrast CT shot through the heart with only one PA. Normally, you might think one PA is just volume averaging - but once you've been shown volume loss on one side your suspicion for this should be raised.

Trivia to know:

- It's seen on the opposite side of the aortic arch (Absent right PA with left sided aortic arch, Absent left PA with right sided aortic arch).
- Associated with PDA
- Interrupted left PA is associated with TOF, and Truncus

Pneumonia

Bacterial Infection		
Strep Pneumo	Lobar Consolidation	Favors lower lobes. Can be severe in sickle cell patients post splenectomy. The most common cause of pneumonia in AIDS patient.
Staph A.	Bronchopneumonia - patchy opacities	Often bilateral, and can make abscess. Can be spread via the blood in endocarditis patients
Anthrax	Hemorrhagic lymphadenitis, mediastinitis, and hemothorax	Classic Look: Mediastinal widening with pleural effusion in the setting of bio-terrorism
Klebsiella	Buzzword: “Bulging Fissure” from exuberant inflammation. More likely to have pleural effusions, empyema, and cavity than conventional pneumonia.	Alcoholic and Nursing Home Patients. Step 1 Buzzword was “currant jelly sputum”
H. Flu	Usually bronchitis, sometimes bilateral lower lobe bronchopneumonia	Seen in COPDs , and people without a spleen
Pseudomonas	Patchy opacities, with abscess formation	CUers on a ventilator (also CF and Primary Ciliary Dyskinesia). Pleural effusions are common. Hit usually small
Legionella	Peripheral and sublobar airspace opacity	Seen in COPDs , and around crappy air conditioners. Only cavitates in immunosuppressed patients. X-ray tends to lag behind resolution of symptoms.
Aspiration	Aerobes, with airspace opacities. They can cavitate, and abscess is not uncommon	Posterior lobes if supine when aspirating, Basal ^lower lobes in upright aspiration May favor the right side, just like an ET tube. The most common complication is empyema (which can get a bronchopleural fistula).
Actinomyces	Airspace in peripheral lower lobes. Can be aggressive and cause rib osteomyelitis/ invade adjacent chest wall.	Classic story is dental procedure gone bad, leading to mandible osteo, leading to aspiration.
Mycoplasma	• Fine reticular pattern on CXR, Patchy airspace opacity with tree-in-bud	

Immunocompromised

Post Bone Marrow Transplant: You see pulmonary infections in nearly 50% of people after bone marrow transplant, and this is often listed as the most common cause of death in this population. The findings are segregated into: early neutropenic, early, and late - and often tested as such.

Post Bone Marrow Transplant (Pulmonary Findings)		
Early Neutropenic (0-30)	Early (30-90)	Late > 90
Pulmonary Edema, Hemorrhage, Drug Induced Lung Injury	PCP, CMY	Bronchiolitis Obliterans, Cryptogenic Organizing Pneumonia
Fungal Pneumonia (invasive aspergillosis)		

Post Bone Marrow Transplant Graft vs Host	
Acute (20-100 Days)	Chronic (> 100 days)
Favors extrapulmonary systems (skin, liver, GI tract)	Lymphocytic Infiltration of the airways and obliterative bronchiolitis.

AIDS Related Pulmonary Infection:

Questions related to AIDS and pulmonary infection are typically written in one of two ways (1) with regard to the CD4 count, and (2) by showing you a very characteristic infection.

Infections in AIDS by CD4		CT Pattern-With AIDS	
>200	Bacterial Infections, TB	Focal Airspace Opacity	Bacterial Infection (Strep Pneumonia) is the most common. DDx should include TB if low CD4. If it's a chronic opacity think Lymphoma or Kaposi.
<200	PCP, Atypical Mycobacterial	Multi-Focal Airspace Opacity	Bacterial, or Fungal
< 100	CMV, Disseminated Fungal, Mycobacterial	Ground Glass	This is gonna be PCP (if that's not a choice it could be CMV if CD4 is <100).

PCP: This is the most classic AIDS infection. This is the one they are most likely to show you. **Ground glass opacity** is the dominant finding, and is seen bilaterally in the perihilar regions with sparing of the lung periphery. Cysts, which are usually thin walled, can occur in the ground glass opacities about 30% of the time.

AIDS High Yield Trivia / Buzzwords:

- * Most common airspace opacity = Strep Pneumonia
- * If they show you a CT with ground glass = PCP
- * "Flame Shaped" Perihilar opacity = Kaposi Sarcoma
- * Persistent Opacities = Lymphoma
- * Lung Cysts = LIP
- * Lungs Cysts + Ground Glass + Pneumothorax = PCP
- * Hypervascular Lymph Nodes = Castleman's or Kaposi

TB

You can think about TB as either (a) Primary, (b) Primary Progressive, (c) Latent or (d) Post Primary / Reactivation.

- **Primary:** Essentially you inhaled the bug, and it causes necrosis. Your body attacks and forms a granuloma (Ghon Focus). You can end up with nodal expansion (which is bulky in kids, and less common in adults), this can calcify and you get a “Ranke Complex.” The bulky nodes can actually cause compression leading to atelectasis (which is often lobar). If the node ruptures you can end up with either (a) endobronchial spread or (b) hematogenous spread - depending on if the rupture is into the bronchus or a vessel. This hematogenous spread manifests as a miliary pattern. **Cavitation in the primary setting is NOT common.** Effusions can be seen but are more common in adults (uncommon in kids).
- **Primary' Progressive:** This term refers to local progression of parenchymal disease with the **development of cavitation** (at the initial site of infection / or hematogenous spread). This primary progression is uncommon - with the main risk factor being **HIV**. Other risk factors are all the things that make you immunosuppressed - transplant patients, people on steroids. The ones you might not think about is jejunioileal bypass, subtotal gastrectomy, and silicosis. This form is **similar in course to post primary disease**.
- **Latent:** This is a positive PPD, with a negative CXR, and no symptoms. If you got the TB vaccine, you are considered latent if your PPD converts by the US health care system/industry. This scenario buys you 9 months of INH and maybe some nice drug induced hepatitis. •
- **Post Primary (reactivation):** This happens about 5% of the time, and describes an endogenous reactivation of a latent infection. The classic location is in the apical and posterior upper lobe and superior lower lobe (more oxygen, less lymphatics). In primary infection you tend to have healing. In post primary infection you tend to have progression. The **development of a cavity** is the thing to look for when you want to call this. Arteries near the cavity can get all pseudoaneurysm'd up - “Rasmussen Aneurysm” they call it - in the setting of a TB cavity.

Immune Reconstitution Inflammatory Syndrome: The story will be patient with TB and AIDS started on highly active anti-retroviral therapy (HAART) now doing worse. The therapy is steroids.

Pleural Involvement with TB: This can occur at any time at the initial infection. In primary TB the development of a pleural effusion can be seen around 3-6 months after infection - as a hypersensitivity response. This pleural fluid is usually culture negative (usually in this case is like 60%). You have to actually biopsy the pleura to increase your diagnostic yield. You don't see pleural effusions as much with post primary disease, but when you do, the fluid is usually culture positive.

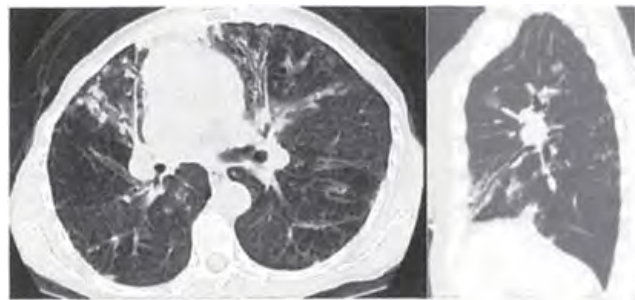
High Yield Factoids Regarding TB:

- * Primary = No Cavity, Post Primary / Primary Progressive = Cavity
- # Ghon Lesion = Calcified TB Granuloma ; sequela of primary TB
- Ranke Complex = Calcified TB Granuloma + Calcified Hilar Node ; Healed primary TB
- Bulky Hilar and Paratracheal Adenopathy = Kids
- * Location for Reactivation TB = Posterior / Apical upper lobes, Superior Lower Lobes
- Miliary Spread when? - Hematogenous dissemination (usually in the setting of reactivation), but can be in primary progressive TB as well
- * Reactive TB Pattern (Cavitation) seen in HIV patient when the CD4 is > 200
- * Primary Progressive Pattern (Adenopathy, Consolidation, Miliary Spread) in HIV is $CD4 < 200$
- TB does NOT usually cause a lobar pattern in HIV

Non Tuberculous Mycobacteria: Not all mycobacterium is TB. The two non-TB forms worth knowing are mycobacterium avium-intracellulare complex (MAC) and Mycobacterium Kansasii. I find that grouping these things into 4 buckets is most useful for understanding and remembering them.

- **Cavitary** (“Classic”) - This one is usually caused by MAC. It favors an old white man with COPD (or other chronic lung disease), and it looks like reactivation TB. So you have an upper lobe cavitary lesion with adjacent nodules (suggesting endobronchial spread).

- * **Bronchiectatic** (“Non-Classic”)
 - This is the so called “**Lady Windermere**” disease (everyone knows it’s just not lady-like to cough). They often do not cough, and are asymptomatic. This favors an old white lady. You see tree-in-bud opacities and cylindric bronchiectasis in the right middle lobe and lingula.



Lady Windermere - MAC
- Bronchiectasis in right middle lobe and lingula

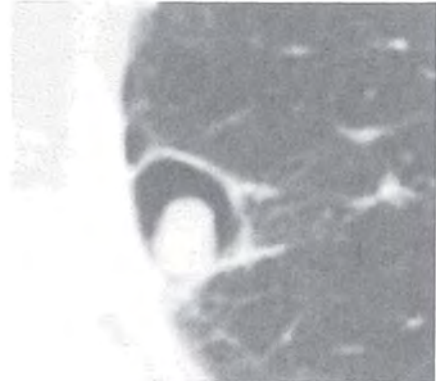
- **HIV Patients** - You see this with low CD4s (< 100). The idea is that it’s a GI infection disseminated in the blood. You get a big spleen and liver. It frequently is mixed with other pulmonary infections (PCP, etc...) given the low CD4 - so the lungs can look like anything. Mediastinal lymphadenopathy is the most common manifestation.
- * **Hypersensitivity Pneumonitis** - This is the so called “**hot-tub lung.**” Where you get aerosolized bugs (which exist in natural sea water and in fresh water). The lungs look like ill-defined, **ground glass centrilobular nodules**.

Non Tuberculous Mycobacteria - Rapid Review		
Cavitary Type	Old White Male Smoker	Looks like reactivation TB
Non-Classic (<i>Lady Windermere</i>)	Old Lady	Middle Lobe and Lingula, bronchiectasis and tree in bud.
HIV	Low CD4 (< 100)	Mediastinal Lymphadenopathy
Hypersensitivity (<i>Hot Tub Lung</i>)	History of hot tub use	Ground glass centrilobular nodules

Fungal

Aspergillus: So this can cause a variable appearance and the trivia surrounding that variability comes in three flavors: (1) normal immune, (2) immune depressed, or (3) Hyper-immune.

- *Normal Immune:* This is the situation when aspergillus makes a fungus ball “Aspergilloma” in an existing cavity. The way this is asked is pretty much always the same. They will show you a fungus ball, and they want you to call it invasive. Don’t fall for that. This is not the same thing as invasive. They can be totally normal people who have a cavity from trauma, or prior infection ect...



Aspergilloma or Fungus Ball

Normal Immune Patient with Fungus in premade cavity

- * *Immune Suppressed (AIDS, or Transplant Patient):* This is when you get your **invasive aspergillus**. This is going to be shown one of two ways. (1) A **halo sign** - consolidative nodule/mass with a ground glass halo. The halo of ground glass is actually the invasive component. (2) **Air Crescent sign** - a thin crescent of air within the consolidative mass. This actually represents healing, as the necrotic lung separates from the parenchyma. The timing is usually about 2-3 weeks after treatment. Lastly, they could show you some peripheral wedge shaped infarcts in the setting of some halo signs.

- * *Hyper-Immune:* This is your asthmatic with **ABPA**. Allergic Brocho-Pulmonary Aspergillosis. This is “Always” seen in patients with **long standing asthma** (sometimes CF). You classically have upper lobe central saccular bronchiectasis with mucoid impaction (finger-in-glove).

“Gloved Finger Sign” -A branching tube or finger like opacity extending from the hila, representing an impacted mucous filled bronchus. It’s nonspecific, and can be seen with cancer, endobronchial lesions, bronchial atresia, etc... but if they show you this the answer is going to be ABPA.

Mucormycosis - This aggressive fungal infection almost always occurs in impaired patients (AIDS, Steroids, Bad Diabetics Etc..). You usually think about mucor eating some diabetic’s face off, but it can also occur in the lungs. Think about this when you have invasion of the mediastinum, pleura, and chest wall.

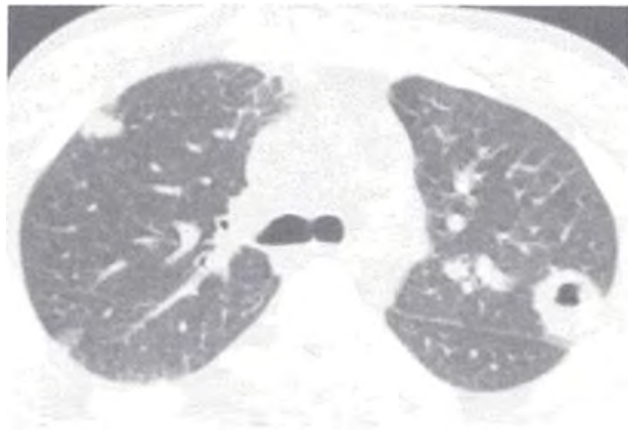
Viral

CMV - This can be seen in two classic scenarios: (1) Reactivation of the latent virus after prolonged immunosuppression (post bone marrow transplant), and (2) Infusing of the CMV positive marrow or in other blood products. The timing for bone marrow patients is “early” between 30-90 days. The radiographic appearance is multiple nodules, ground glass or consolidative.

Random Viral Trivia		
Measles	Multifocal ground glass opacities with small nodular opacities	Pneumonia can be before or after the skin lesions. Complications higher in pregnant and immunocompromised
Influenza	Coalescent lower lobe opacity. Pleural effusion is rare.	
SARS	Lower lobe predominant ground glass opacities	
Varicella	Multiple peripheral nodular opacities. They form small round calcific lung nodules in the healed version.	About 1/6 th with skin findings will get a pneumonia. It's usually in kids. When you see it they are usually immunocompromised (have AIDS or lymphoma).
Ebstein Barr	Uncommonly affects the lung. Can cause lymph node enlargement	Most common radiographic abnormality is a big spleen.

Septic Emboli

There are a variety of ways you can throw infectious material into the lungs via the bloodstream (pulmonary arteries). Some common sources would include; infected tricuspid valves, infection in the body, infected catheters, infected teeth... etc...



Septic Emboli - *Multiple round opacities, one with cavitation*

Things to know about Septic Emboli:

- * It's lower lobe predominant (more blood flow)
- * You get peripheral nodular densities and wedge shaped densities (can infarct).
- * They can **cavitate**, and likely will be cavitated if they show you a CT image.
- * The **feeding vessel sign** - nodule with a big vessel going into it can be shown (also seen with hematogenous mets).
- * Empyema and pneumothorax are both known complications.

CAVITY Mnemonic For Lung Cavity:

- C - Cancer (usually squamous cell)
- A - Auto-immune (Wegeners, Rheumatoid / Caplan Syndrome)
- V - Vascular - Septic Emboli / Bland Emboli
- I - Infection - TB
- T - Trauma - Pneumatocoles
- Y - Young - "Congenital" - CCAMs, Sequestrations

Lemierre Syndrome: This is an eponym referring to **jugular vein thrombosis with septic emboli** classically seen after an oropharyngeal infection or recent ENT surgery.

Classic Lemierre Question:

Q: What is the bacterial agent responsible in the majority of cases?

*A: "**Fusobacterium Necrophorum.**"*

Lung Cancer

Screening: Recently, the US preventive services task force has approved lung cancer screening with low dose CT for asymptomatic adults aged 55-80 who have a 30 pack-year history and currently smoke (or have quit within the past 15 years). Obviously this is going to be a huge cash cow for radiology not just in the CT, but for the numerous incidental follow ups. Follow up recommendations are still being developed (so they won't be on the CORE). The old Fleischner society stuff might be, with the most likely questions being that *Fleischner Society Recommendations do NOT apply to patient's with known cancers.*

Solitary Pulmonary Nodule: A SPN is defined as a round or oval lesion measuring less than 3cm in diameter (more than 3cm = mass). Technically to be "solitary" it needs to be surrounded by lung parenchyma, with no associated adenopathy, or pleural effusion.

There are 4 classic "benign calcification" patterns: solid, laminated, central, and popcorn. Anything else is considered suspicious. Eccentric patterns are considered the most suspicious. Some notable (testable) exceptions include when you see popcorn and central calcifications in the setting of a GI cancer. Solid calcifications can be bad in the setting of osteosarcoma.

Solid/Diffuse

Laminated

Central

Popcorn

Nodule Qualities That Make You Think It's B9	Nodule Qualities That Make You Think It's Cancer
Presence of Fat	Spiculated Margins "Corona Radiata Sign"
Rapid Doubling Time (less than 1 month)	Air Bronchogram through the nodules (usually Adenocarcinoma in situ)
Slow Doubling Time (longer than 16 months) * Stable at two years = B9	Partially solid lesions with ground glass component

Solid and Ground Glass Components: A **part solid lesion with a ground glass component is the most suspicious morphology you can have.** Non-solids (only ground glass) is intermediate. Totally solid is actually the least likely morphology to be cancer.

PET for SPN: You can use PET for SPNs larger than 1cm. Lung Cancer is supposed to be HOT (SUR > 2.5). Having said that, infectious and granulomatous nodules can also be hot. If you are dealing with a ground glass nodule it's more likely to be:

COLD = Cancer, HOT = Infection.

Lung Cancer Risk factors include; being over 30 (under 30 is super rare), exposures to bad stuff (arsenic, nickel, asbestos, chromium, beryllium, radon), having lung fibrosis, COPD (even if you didn't smoke), and family history.

Types: There 4 types of lung cancer.

- * **Squamous:** Usually centrally located, and strongly associated with smoking. Cavitation is common, and is the most likely question. The prognosis is relatively good, as this subtype likes to met late. You can get ectopic PTH production.
- * **Small Cell:** Usually central (common near the main or lobar bronchi). You may only have central lymphadenopathy with this one. It has a terrible prognosis, and is basically a death sentence. Paraneoplastic syndromes with SIADH can occur.

Lambert Eaton: Paraneoplastic Syndrome seen with patient's having Small Cell Lung Cancer. They get proximal weakness from abnormal release of acetylcholine at the neuromuscular junction. The clinical presentation often comes before the cancer diagnosis.
- * **Large Cell:** Usually peripheral and large (> 4cm). Prognosis sucks.
- * **Adenocarcinoma:** It's usually peripheral (75%) often in the upper lobes. It's the most common subtype overall and the **most common subtype to present as a solitary pulmonary nodule**. This guy has a known association with lung fibrosis.

BAC (Subtype of Adenocarcinoma): This has recently undergone a name change. Why change its name? Well it's a simple reason. Academic Radiologists need to be on committees to get promoted. Committees need an excuse to go on vacation ("International Meetings" they call them). Name changes happen...

- * **Atypical Adenomatous Hyperplasia of Lung (AAH):** This is a precursor of adenocarcinoma of the lung.
- * **Adenocarcinoma in situ (ACIS):** These are < 3cm. This in itself has multiple subtypes. The most common of these is the non-mucinous one. *
- * **Minimally Invasive Adenocarcinoma (MIA):** These are also < 3cm. The distinction is that there is < 5mm of stromal invasion (> 5mm will be called a lepidic predominant adeoncarcinoma).

o Things to know:

It's usually ground glass

It's classically COLD on PET

"Fried Egg" - Ground glass halo around nodule

"Pseudocavitation" bubble like lucencies

Key Concept: The larger the solid component of the "part solid" nodule gets the more likely it is to be malignant. In other words, **partially solid nodules are more likely to be cancer than ground glass nodules.**



Staging:

Lung cancer staging is different for small cell vs non-small cell (NSCLC). NSCLC staging is much more testable. *The big thing to know about lung cancer staging is that stage 3B is unresectable.* So what makes something stage 3B?

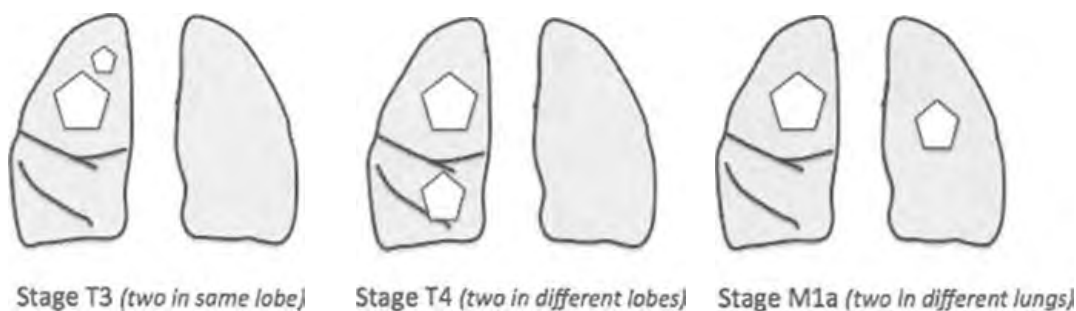
Stage 3B (NOT resectable)

Supraclavicular, Scalene or Contralateral
Mediastinal or Hilar Adenopathy

Tumor in the same lung but different lobes from
the primary mass

Malignant Pleural Effusion

Another piece of trivia I think lends well to multiple choice, is the difference in multi-centric locations of tumors. Two in the same lobe is a T3, Two in different ipsilateral lobes is T4, and Two in different lungs is an M1a.

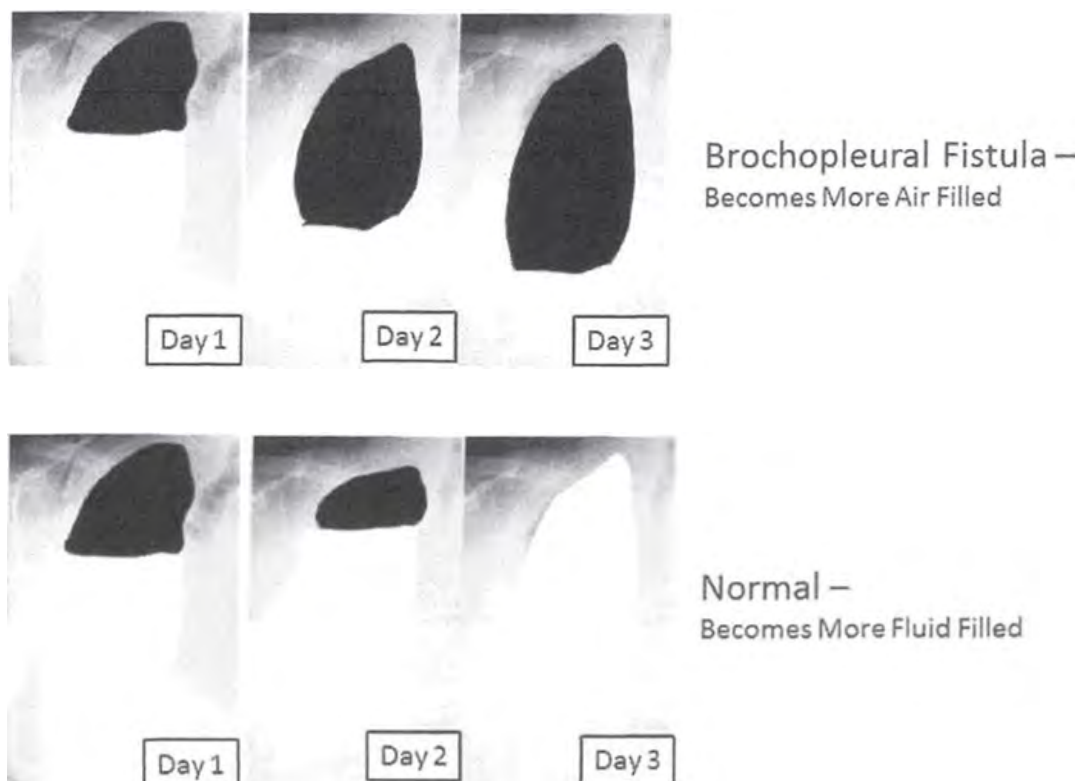


Treatment: Treatment for lung cancer is either surgery, radiotherapy, chemotherapy, thermal ablation (RFA/MWA) in isolation or combination, depending on the stage. **Stage 3B being non-operable as the big testable point.**

Radiation Changes: - The appearance of radiation pneumonitis is variable and based on the volume of lung involved, how much/long radiation was given, and if chemotherapy was administered as well.

Radiation Changes	
Early (<i>within 1-3 months</i>)	Late
Homogenous or patchy ground glass opacities.	Dense consolidation, traction bronchiectasis, and volume loss.

Bronchopleural Fistula - This is an uncommon complication of pneumonectomy, that has a characteristic look and therefore easy to test. So normally after a pneumonectomy the space will fill with fluid. If you see it filling with air than this is the dead give away. You can confirm the diagnosis with a xenon nuclear medicine ventilation study, which will show-xenon in the pneumonectomy space. The major risk factor is ischemia to the bronchi (disrupted blood supply from aggressive lymph node dissection, or using a long bronchial stump).



Other Tumors:

Mets - Metastatic disease to the lungs can be thought of in 3 categories; direct invasion, hematogenous, lymphangitic:

- * **Direct Invasion:** This is seen with cancer of the mediastinum, pleura, or chest wall. The most common situation is an esophageal carcinoma, lymphoma, or malignant germ cell tumor. More rarely you are going to have mets to the pleura then invading the lung. Even more rarely you can have malignant mesothelioma, which can invade the lung. It should be obvious.

Feeding Vessel Sign: A prominent pulmonary vessel heading into a nodule. This supposedly means it's from a hematogenous origin. It's nonspecific - but if you see it the answer is (1) mets , or (2) septic emboli.
- * **Hematogenous Mets:** The most common manifestation of hematogenous mets to the lung is the pulmonary nodule (usually multiple, in a **random distribution**, and **favoring the lower lobes** which have greater blood volume). The nodules tend to be smoother than the primary neoplasm. The main culprits are breast, kidney, thyroid, colon, and head & neck squamous cells. Obviously the squamous mets can cavitate. **“Cannonball Mets” are classically from renal cell or choriocarcinoma** (testicle).
- * **Lymphangitic Carcinomatosis (LC):** The most common cause of unilateral LC is actually bronchogenic carcinoma lung cancer invading the lymphatics. The most common extrathoracic culprits are breast, stomach, pancreas, and prostate. The finding is nodular thickening of the interlobular septa and subpleural interstitium. Unlike interstitial fibrosis, this thickening classically does NOT distort the pulmonary lobule.

Carcinoid: Carcinoid can be classified either based on location; (a) peripheral pulmonary, and (b) bronchial - or by histologic type (a) typical and (b) atypical. The typical carcinoids are slow growing and locally invasive (only met to nodes about 10% of the time). There is no association with smoking. These typical carcinoids typically **appear centrally within a bronchus** (only 1% are in the trachea). As they occur endobronchially, they **often cause obstructive symptoms**. They can also cause hemoptysis because they are highly vascular. An octreotide scan can be used to localize a carcinoid tumor. The pulmonary tumors can cause a carcinoid syndrome with flushing etc... The valvular degradation that occurs tends to be on the left side (mitral and aortic), as opposed to the GI carcinoid syndrome which affects the right side (tricuspid and pulmonic). The atypical carcinoids are more rare, seen in older patients, and more likely to be a mass.

Adenoid Cystic (Cylindroma) - This is the most common bronchial gland tumor. It is NOT associated with smoking. They are usually in the main or lobar bronchus.

Carcinoid	More common in bronchus, Rare in Trachea
Adenoid Cystic	Occurs in Bronchus, 20:1 more common than carcinoid in the Trachea.

Lymphoma - There are basically 4 flavors of pulmonary lymphoma; primary, secondary, AIDS related, or PTLT. Radiographic patterns are variable and can be lymphagitic spread (uncommon), parahilar airspace opacities, and/or mediastinal adenopathy.

- * **Primary:** This is rare, and usually non-Hodgkin in subtype. You define it as the lack of extrathoracic involvement for 3 months. Almost always (80%) of the time we are talking about a low grade MALToma.
- * **Secondary:** Here we are talking about pulmonary involvement of a systemic lymphoma. This is much much much more common than primary lung lymphoma. The thing to see is that NHL is much more common, but if you have HL it is more likely to involve the lungs. With HL you get nodes and parenchyma, in NHL you might just get parenchyma. *

Secondary NHL	Secondary HL
80-90% of lymphoma cases	10-20% of lymphoma cases
45% have intrathoracic disease at presentation	85% have intrathoracic disease at presentation
25% have pulmonary parenchymal disease	40% have pulmonary parenchymal disease
Pulmonary involvement frequently occurs in the absence of mediastinal disease	Lung involvement almost always associated with intrathoracic lymph node enlargement

- * **PTLD:** This is seen after solid organ or stem cell transplant. **This usually occurs within a year of transplant** (late presentations > 1 year have a more aggressive course). This is a B-Cell lymphoma, with a relationship with EB Virus. You can have both nodal and extra nodal disease. The typical look is well defined pulmonary nodules / mass, patchy airspace consolidation, halo sign, and interlobular septal thickening.

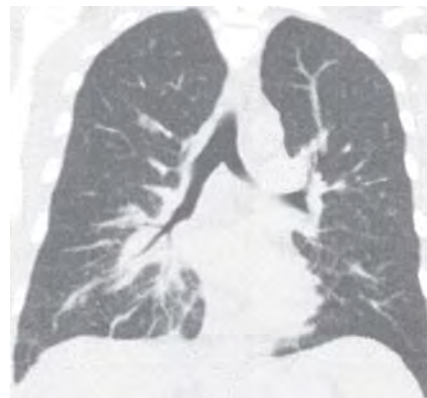
- **AIDS related pulmonary lymphoma (ARL)** - This is the second most common lung tumor in AIDS patients (Kaposi's is first). Almost exclusively a high grade NHL. There is a relationship with EBV. It is seen in patients with a **CD4 < 100**. The presentation is still variable with multiple peripheral nodules ranging from 1cm-5cm being considered the most common manifestation. Extranodal locations (CNS, bone marrow, lung, liver, bowel) is common. **AIDS patient with lung nodules, pleural effusion, and lymphadenopathy = Lymphoma.**

Kaposi Sarcoma: This is the most common lung tumor in AIDS patients (Lymphoma is number two). The tracheobronchial mucosa and perihilar lung are favored. The buzzword is "flame shaped." A bloody pleural effusion is common (50%).

Kaposi Sarcoma	Lymphoma
Thallium ²⁰¹ Positive	Thallium ²⁰¹ Positive
Gallium ⁶⁷ Negative	Gallium ⁶⁷ Positive

Things to know about KS:

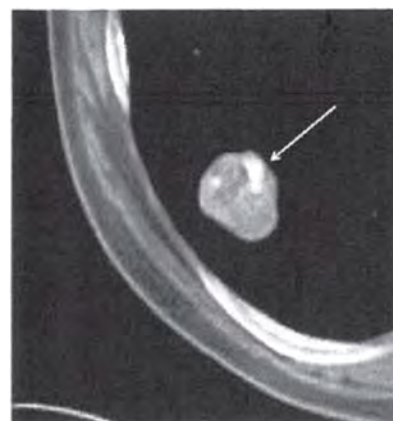
- * *Most common lung tumor in AIDS (requires CD4 < 200)*
- * *Most common hepatic neoplasm in AIDS*
- * *Buzzword = Flame Shaped Opacities*
- * *Slow Growth, with asymptomatic patients (despite lungs looking terrible)*
- * *Thallium Positive, Gallium Negative*



Kaposi Sarcoma
- "Flame Shaped" Hilar Opacities

Hamartoma - this is an Aunt Minnie because it will have **macroscopic fat and "popcorn" calcifications**. It is the most common benign lung mass. It's usually incidental, but can cause problems if it's endobronchial (rare - like 2%).

Technically the fat is only seen in 60%, but for sure if the CORE shows it, it will have fat. These **can be hot on PET**, they are still benign.



Pulmonary Hamartoma

- Popcorn Calcifications
- Fat Density

Congenital:

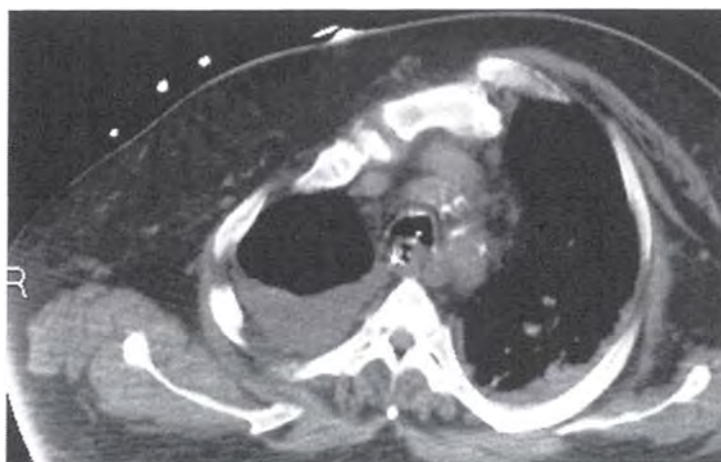
Bronchial Atresia - This most commonly involves the apico-posterior segment of the left upper lobe. The usual look is a blind ending bronchus, filled with mucus (**“finger in glove”**), with the **distal lung hyper-inflated** - from collateral drift and air trapping.

AVM - They can occur sporadically. For the purpose of multiple choice when you see them think about HHT (Hereditary Hemorrhagic Telangiectasia / Osier Weber Rendu). Pulmonary AVMs are most commonly found in the lower lobes (more blood flow), and can be a source of right to left shunt (**worry about stroke and brain abscess**). The rule of **treating once the afferent vessel is 3mm** is based on some tiny little abstract and not powered at all. Having said that, it's quoted all the time, and a frequent source of trivia that is easily tested.

Persistent Left SVC - This is the most common congenital venous anomaly of the chest. It usually only matters when the medicine guys drop a line in it on the floor and it causes a confusing post CXR (line is in a left paramedian location). It usually **drains into the coronary sinus**. In a minority of cases (like 5%) it will drain into the left atrium, and cause right to left shunt physiology (very mild though). This is typically shown on an axial CT at the level of the AP window, or with a pacemaker (or line) going into the right heart from the left.

Swyer-James - This is the classic **unilateral lucent lung**. It typically occurs after a viral lung infection in childhood resulting in **post infectious obliterative bronchiolitis** (from constrictive bronchiolitis). The *size of the affected lobe is smaller* than a normal lobe (it's not hyperexpanded).

Poland - Unilateral absence of a pectoral muscle. It can cause a unilateral hyperlucent chest. They can have limb issues (small weird arms / hands).



Poland Syndrome - Absent Pectoral on Right

Sequestration These are grouped into intralobar and extralobar with the distinction being which has a pleural covering. You can NOT tell the difference radiographically. The practical difference is age of presentation; intralobar presents in adolescence or adulthood with recurrent pneumonias, extralobar presents in infancy with respiratory compromise.

Intralobar: Much more common (75%). Presents in adolescence or adulthood as recurrent pneumonias (bacteria migrate in from pores of Kohn). **Most commonly in the left lower lobe posterior segment (2/3s)**. Uncommon in the upper lobes. In contradistinction from extralobar sequestration, it is rarely associated with other developmental abnormalities.

Extralobar: Less common of the two (25%). Presents in infancy with respiratory compromise (primarily because of the associated anomalies - *Congenital cystic adenomatoid malformation (CCAM)*, *congenital diaphragmatic hernia*, *vertebral anomalies*, *congenital heart disease*, *pulmonary Hypoplasia*). It rarely gets infected since it has its own pleural covering.

Intralobar	Extralobar
More Common	Less Common
Presents in Adolescence	Presents in Infancy
Recurrent Infections	Associated Congenital Anomalies
No Pleural Cover	Has it's own pleural cover

CCAM - As the name suggests it's a malformation of adenomatoid stuff that replaces normal lung. Most of the time it only affects one lobe. There is no lobar preference (unlike CLE which favors the left upper lobe). There are cystic and solid types (type 1 cystic, type 3 solid, type 2 in the middle). There is a crop of knuckle heads who want to call these things CPAMs and have 5 types, which I'm sure is evidence based and will really make an impact in the way these things are treated. CCAMs communicate with the airway, and therefore fill with air. Most of these things (like 90%) will spontaneously decrease in size in the third trimester. The treatment (*at least in the US*) is to cut these things out, because of the iddy bitty theoretical risk of malignant transformation (*pleuropulmonary blastoma*, *rhabdomyosarcoma*).

Diseases Primarily Involving the Interstitial Lung

Cystic Lung Disease:

Pulmonary Langerhans Cell Histiocytosis (LCH) - This cystic lung disease classically effects **smokers, who are young (20s-30s)**. The disease starts out with centrilobular nodules with an upper lobe predominance. These nodules eventually cavitate into cysts which are thin walled to start, and then some become more thick walled. Late in the disease you are primarily seeing cysts. The buzzword is

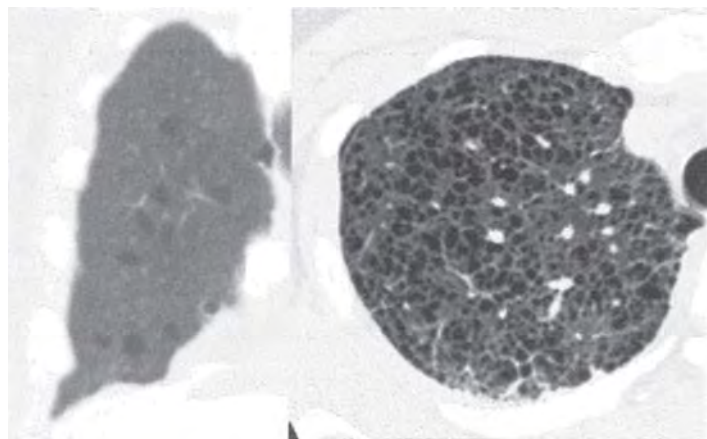
bizarre shaped, which occurs when 2 or more cysts merge together. In about half the cases this spontaneously resolves (especially if you stop smoking). Another piece of trivia is the LCH spares the costophrenic angles.

What Sparing the Costophrenic Angles???
LCH and Hypersensitivity Pneumonitis

Lymphangiomyomatosis (LAM) - This cystic lung disease can occur in **child bearing aged women** or in association with **tuberous sclerosis (a trick is to show the kidneys with multiple AMLs first)**. The cysts are **thinned walled** with a uniform distribution. There is an **association with chylothorax** (which is HIGH YIELD Trivia). The pathophysiology is that it is estrogen dependent (why it strongly favors women). This is usually progressive despite attempts at hormonal therapy (tamoxifen).

LCH	LAM	LCH	Bizarre Shape	Thick Wall
Cysts and Nodules	Cysts (no nodules)			
Smoker	Women, Pts with Tuberous Sclerosis	LAM	Round	Thin Wall
Upper and Mid Lungs	Diffuse	BHD	Oval	Thin Wall
Thicker Cysts (Bizarre)	Thin Round Cysts			

Birt Hogg Dube (BHD) - This is a **total zebra**. This cystic lung disease has **thin walled “oval” shaped cysts**. There is an association with renal findings (bilateral oncocytomas, and chromophobe RCCs). They also have a bunch of gross skin stuff.



Birt-Hogg-Dube
- Oval Cysts

LAM
~ Multiple Thin Walled Round Cysts

Lymphocytic Interstitial Pneumonitis (LIP) - This is a benign lymphoproliferative disorder, with infiltration of the lungs. It has an association with autoimmune diseases (**SLE, RA, Sjogrens**). **The big one to know is Sjogrens which is concomitant in 25% of LIP cases.** The other one to know is **HIV** - which is the LIP in a younger patient (*children, - LIP in HIV positive adults is rare*). There is also an association with Castleman's. The appearance of LIP varies depending on the underlying cause. **The cystic lung disease is usually thin walled, "deep within the lung parenchyma," and seen predominantly with Sjogrens.** The dominant feature described as ground glass or nodules is seen more in the other causes and is far beyond the scope of the CORE exam.

When I say LIP... You say Sjogrens & HIV

When I say LIP in a kid... You say HIV

Pneumocystis Pneumonia (PCP) - This is the most common opportunistic infection in AIDS. The typical buzzword is **ground glass appearance, predominantly in the hilar and mid lung zones.** Pneumatocoles are present in 30% of cases. In patients receiving aerosolized prophylaxis, a cystic form is more common, which **may have bilateral thin walled upper lung predominant cysts.** ⁶⁷ **Gallium scan will show diffuse uptake** (Thallium will be negative).

When I say AIDS + Ground Glass Lungs.... You say PCP

Emphysema- The textbook definition is "permanent enlargement of the airspaces distal to the terminal bronchioles accompanied by destruction of the alveolar wall without clear fibrosis." What you need to know are (1) the CXR findings and (2) the different types.

- CXR Findings: Until it's really really bad, CXR doesn't have direct signs, but instead has indirect signs. **Flattening of the hemidiaphragms** is regarded as the most reliable sign. The AP diameter increases. The retrosternal clear space becomes larger. There is a paucity of, or pruning of the blood vessels.

* Types:

- o *Centri-lobular*. By far the **most common type**. Common in asymptomatic elderly patients. It has an apical to basal gradient - **favoring the upper zones** of each lobe. It appears as focal lucencies, located centrally within the secondary pulmonary lobule, often with a central dot representing the central bronchovascular bundle. This **central dot sign is a buzzword**. This is the **type of emphysema dominant in smokers**.

- o *Pan-lubular*: In contradistinction to centrilobular this one favors the lower lobes. It also has a more uniform distribution across parts of the secondary pulmonary lobule. The association is with **alpha 1 antitrypsin**. A piece of trivia is the **"Ritalin Lung"** from IV Ritalin use can also cause a pan-lobular appearance. *If they show this it will be in the coronal view on CT to demonstrate the lower lobe predominance*. Patient's will present in their 60s and 70s (unless they smoke - then they present in their 30s). **Smoking accelerates the process**.

- o *Para-septal*: This one is found adjacent to the pleura and septal lines with a peripheral distribution within the secondary pulmonary lobule. The affected lung is almost always sub-pleural, and demonstrates small focal lucencies up to 10mm in size. This looks like honeycombing but is less than 3 bubbles thick.

- Trivia:

- o Saber Sheath Trachea - Diffuse coronal narrowing of the trachea, sparing the extrathoracic portion. This is said to be pathognomonic for COPD.
- o If the Main PA is larger than the Aorta COPD patient have a worse outcome (pulmonary HTN can be caused by emphysema).
- o Surgery to remove bad lung "volume reduction" is sometimes done

Vanishing Lung Syndrome: This is an idiopathic cause of giant bullous emphysema, resulting from avascular necrosis of the lung parenchyma and hyperinflation. It favors the bilateral upper lobes, and is **defined as bullous disease occupying at least one-third of a hemithorax**. The most common demographic is a young man. About 20% of these guys have alpha-1 antitrypsin deficiency.

Compensatory Emphysema (Postpneumectomy Syndrome): There is no obstructive process here. Instead you have hyperexpansion of one lung to compensate for the absence of the other one.

Honeycomb Lung- When I say honeycombing you should say UIP. However, this is seen with a variety of causes of end stage fibrotic lung processes. The cysts are tightly clustered (2-3 rows thick) and subpleural. The walls are often thick.

Pneumoconioses

As a general rule, these are inhaled so they tend to be upper lobe predominant. You can have centilobular nodules (which makes sense for inhalation), or often perilymphatic nodules - which makes a little less sense, but is critical to remember * especially with silicosis and CWP.

Asbestos Exposure: The term “Asbestosis” refers to the changes of pulmonary fibrosis - NOT actual exposure to the disease. The look is very **similar to UIP**, with the presence of **parietal pleural thickening** being the “most important feature” to distinguish between **IPF** and Asbestosis. Obviously, the history of working in a ship yard or finding asbestos bodies in a bronchoalveolar lavage is helpful.

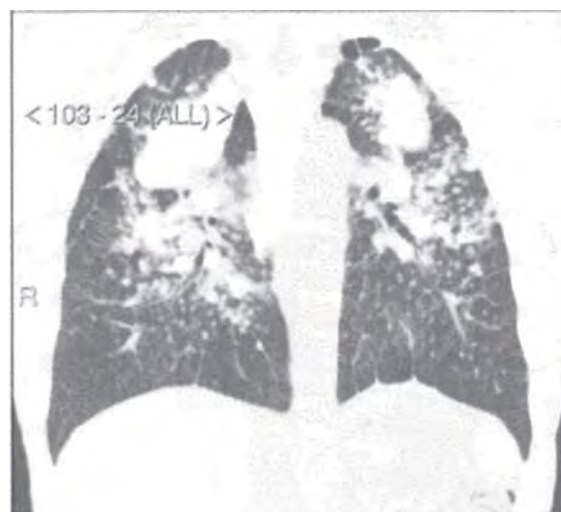
Things to know about Asbestos:

- * “Asbestosis” = the lung fibrosis associated with exposure, NOT actual exposure
- * Interstitial pattern looks like UIP + parietal pleural thickening
- * There is a 20 year latency between initial exposure and development of lung cancer or pleural mesothelioma
- * There is an association with extrapulmonary cancer including: Peritoneal mesothelioma, GI cancer, Renal Cancer, Laryngeal Cancer, and Leukemia
- * Benign pleural effusions are the “earliest pleural based phenomenon ” associated with exposure - still with a lag time of around 5 years

Benign Asbestosis Related Changes: Pleural effusion is the earliest and most common. Pleural plaques may develop around 20-30 years, with calcifications occurring around 40 years. These plaques tend to spare the apices and Costophrenic angles. **Round atelectasis** - which is associated with pleural findings is sometimes called the “asbestos pseudotumor.”

Malignant Mesothelioma - The most common cancer of the pleura. About 80% of them have had asbestos exposure, and development is NOT dose dependent. The lag time is around 30-40 years from exposure. The **buzzword pleural rind** is worth knowing. The tendency is for direct invasion. **Extension into the fissure is highly suggestive.**

Silicosis: This is seen in miners, and quarry workers. You can have simple silicosis, which is going to be multiple nodular opacities favoring the upper lobes, with egg shell calcifications of the hilar nodes. You also get perilymphatic nodules. The complicated type is called **progressive massive fibrosis (PMF)**. **This is the formation of large masses in the upper lobes with radiating strands.** You can see this with both silicosis and coal workers pneumoconiosis (something similar also can happen with Talcosis). These masses can sometimes cavitate - but you should always raise the suspicion of TB when you see this (especially in the setting of silicosis).



Progressive Massive Fibrosis
- Large Apical Masses with Radiating Strands

Silicotuberculosis: Silicosis actually raises your risk of TB by about 3 fold. If you see *cavitation in the setting of silicosis you have to think about TB.*

MRI: Cancer vs PMF	
Cancer = T2 Bright	PMF = T2 Dark

Coal Workers Pneumoconiosis: This is the result of exposure to “washed coal.” Just like silicosis there are simple and complicated forms. There is also an increased risk of TB (just like silicosis). The simple form was multiple nodular opacities, with calcifications showing a central nodular dot. The small nodule pattern tends to have a perilymphatic distribution. The complicated form gives you a progressive massive fibrosis that is similar to that seen in silicosis.

Additional Inhalational Diseases - Not Worthy of a Full Discussion		
Berryliosis	Metal used in aircraft and space industries	Generalized granulomatous disease with hilar adenopathy and upper lobe predominant reticular opacities.
Silo Filler’s Disease	Nitrogen Dioxide	Pulmonary Edema Pattern. Recovery is typically within 5 weeks.
Talcosis	Filler in tablets, sometimes injected (along with drugs) in IV drug users.	Hyperdense micronodules, with conglomerate masses (similar to PMF). Ground glass opacities

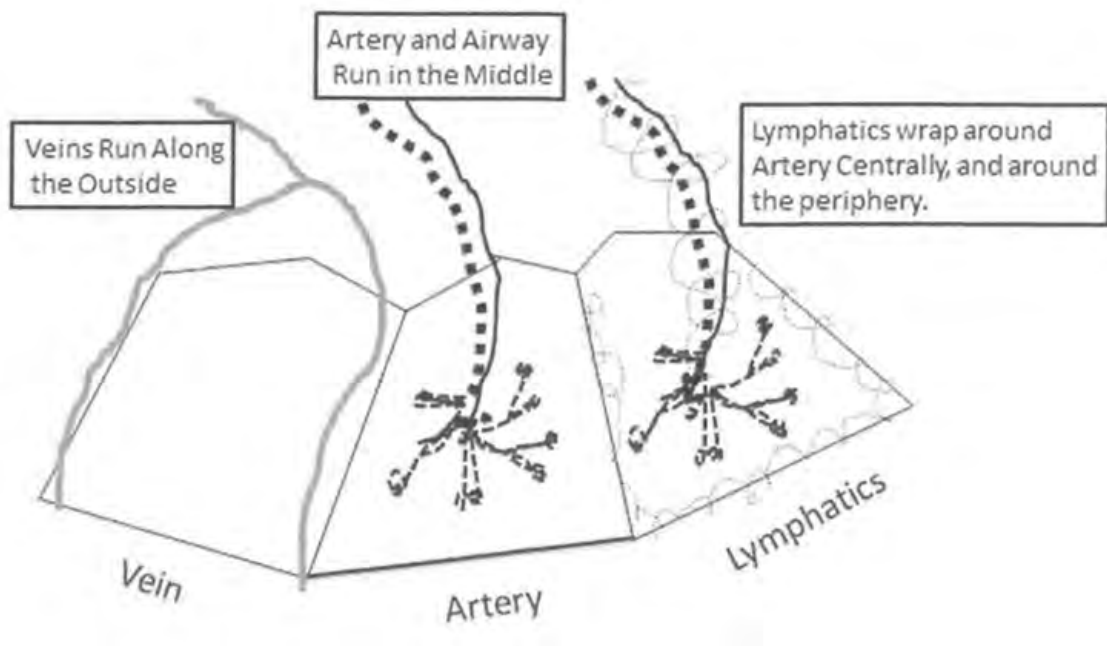
ILDs

Everyone seems to be afraid of interstitial lung diseases. The concept is actually not that complicated, it's just complicated relative to the rest of chest radiology (which overall isn't that complicated). The trick is to ask yourself two main questions: (1) Acute or Chronic ? - as this narrows the differential considerably, and (2) What is the primary finding ? - as this will narrow the differential further. Now, since we are training for the artificial scenario of a multiple choice test (and not the view box), I'll try and keep the focus on superficial trivia, and associations. Remember when you are reading to continue to ask yourself "how can this material be written into a question?"

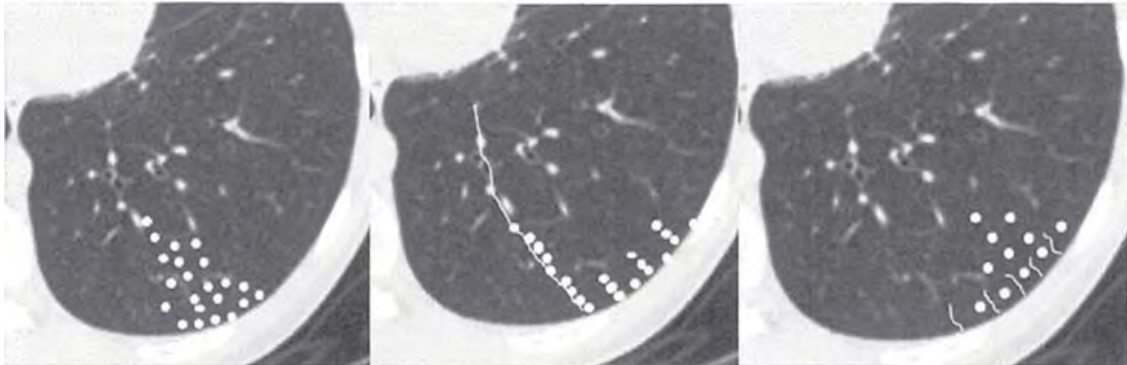
Vocab: Like most of radiology, the bulk of understanding the pathology is knowing the right words to use (plus, big vocabulary makes you sound smart).

- * **Consolidation** = Density that obscures underlying vessels
- * **Ground Glass Opacity** = Density that does **NOT** obscure underlying vessels
- * **Secondary Pulmonary Lobule** = The basic unit of pulmonary structure and function. It is the smallest part of the lung that is surrounded by connective tissue. In the middle runs a terminal bronchial with an accompanying artery. Around the periphery runs the vein and lymphatics. Lymphatics also wrap around the centri lobular artery.

Anatomy of a Secondary Pulmonary Lobule



Nodule Vocabulary (Random, Perilymphatic, Centrilobular)



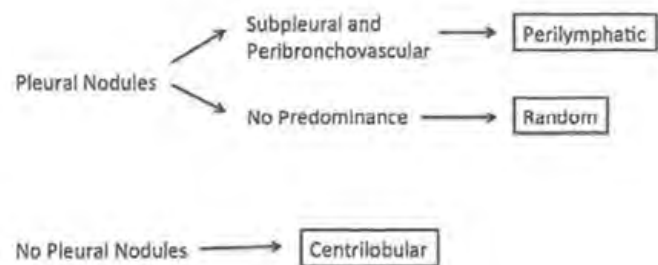
Random

Perilymphatic

Centrilobular

Telling them apart, can be done by first asking if they abut the pleura?

If the answer is no they are centrilobular. If the answer is yes, then ask do they follow a peribronchovascular pattern, if the answer is no then they are random, if the answer if yes then they are perilymphatic.



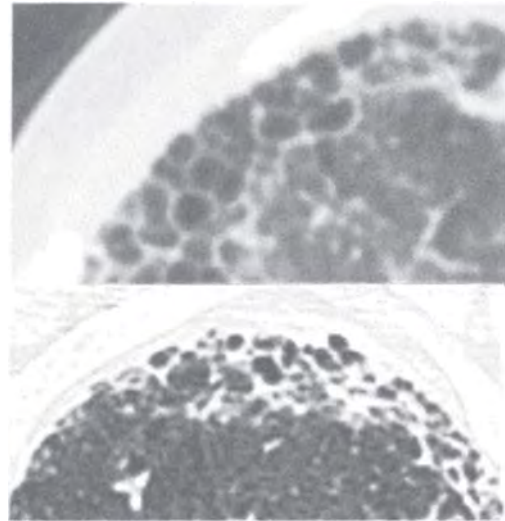
Nodule Pattern	Key DDx
Perilymphatic	<ul style="list-style-type: none"> •Sarcoid (90%), •Lymphangitic Spread of CA •Silicosis
Random	<ul style="list-style-type: none"> •Miliary TB •Mets •Fungal
Centrilobular	<ul style="list-style-type: none"> •Infection •RB-ILD •Hypersensitivity Pneumonitis (<i>if ground glass</i>)

Patterns and Pathology

Interlobular Septal Thickening: Reticular abnormality, that outlines the lobules characteristic shape and size (about 2cm). It's usually from **pulmonary edema** (*usually symmetric and smooth*), or **lymphangitic spread of neoplasm** (*often asymmetric and nodular*). **Kerley B Lines** are the plain film equivalent.

Honeycombing: Cystic areas of lung destruction in a subpleural location. This is a hallmark of UIR Paraseptal emphysema is a mimic, but the distinction is made by how many rows of bubbles.

- *One Row of Bubbles = Paraseptal Emphysema.*
- * *Two-Three Rows of Bubbles = Honeycombing.*



Honeycombing - Two Examples

Pathology Time:

Idiopathic Interstitial Pneumonias - These are NOT diseases, but instead lung reactions to lung injury. They occur in a variety of patterns and variable degrees of inflammation and fibrosis. The causes include: idiopathic, collagen vascular disease, medications, and inhalation.

For practical purposes the answer is either (a) UIP or (b) Not UIP. Not UIP will get better with steroids. UIP will not. UIP has a dismal prognosis (similar to lung cancer). Not UIP often does ok. The CORE exam will likely not make it this simple, and will instead focus on buzzwords, patterns, and associations (which I will now discuss).

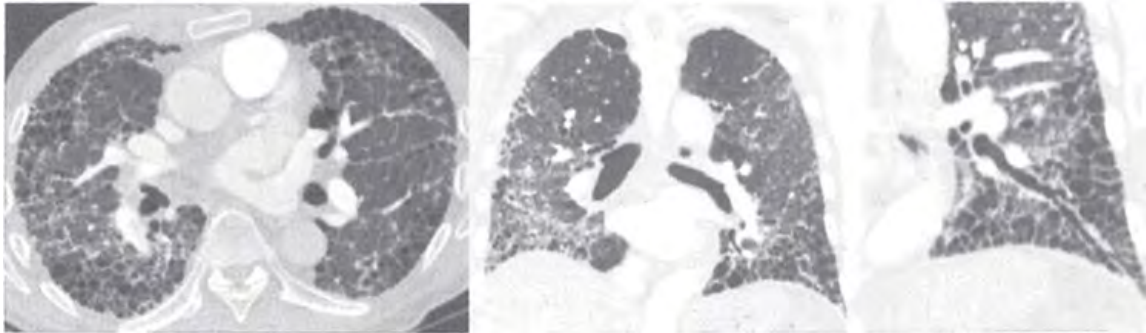
UIP (Usual Interstitial Pneumonia) - This is the most common Interstitial Lung Disease. When the cause is idiopathic it is called IPF. On CXR the lung volume is reduced (duh, it's fibrosis). *Reticular pattern in the posterior costophrenic angle is supposedly the first finding on CXR.*

Buzzwords include:

- **Apical to basal gradient** (it's worse in the lower lobes),
- **Traction bronchiectasis**, and honeycombing.
- **Honeycombing** is found 70% of the time, and people expect you to knee jerk UIP when that term is uttered.
- Histologic Buzzword = Heterogenous. *"The histology was heterogenous"* = **UIP**.

It's important to know that basically any end stage lung disease (be it from sarcoid, RA, Scleroderma, or other collagen vascular disease) has a similar look once the disease has ruined the lungs. **Technically honeycombing is uncommon in end stage sarcoid - but the rest of the lung looks jacked up.*

The prognosis is terrible (similar to lung cancer).



UIP - *Honey Combing, Traction Bronchiectasis, Apical to Basal Gradient*

NSIP (Nonspecific Interstitial Pneumonia)- Less Common than UIP. Even though the name infers that its non-specific, it's actually is a specific entity. Histologically it is homogenous inflammation or fibrosis (UIP was heterogeneous). It is a common pattern in collagen vascular disease, and drug reactions.

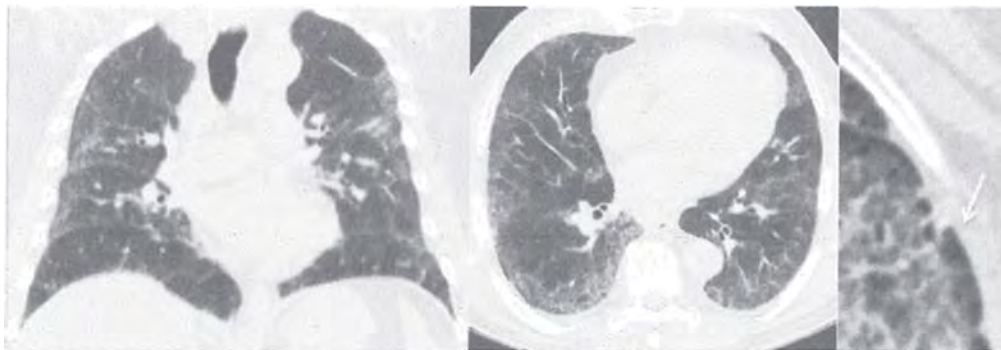
It comes in 2 flavors (cellular or fibrotic):

- Ground Glass Alone = Cellular
- * Ground Glass + Reticulation = Cellular or Fibrotic
- Reticulation + Traction Bronchiectasis = Fibrotic NSIP
- Honeycombing - uncommon and usually minimal in extent

Prognosis:

- * Best = Cellular NSIP
- * OK = Fibrotic NSIP
- * Terrible = UIP/IPF

The disease has a lower lobe, posterior, peripheral predominance with sparing of the immediate subpleural lung seen in up to 50% of cases. This finding of **immediate subpleural sparing** is said to be **highly suggestive**. Ground glass is the NSIP equivalent of honeycombing.



NSIP - *Peripheral Ground Glass with Subpleural Sparing*

UIP	NSIP
Apical to Basal Gradient	Gradient is less obvious (but still more in lower lobes)
Heterogenous Histology	Homogenous Histology
Honeycombing	Ground Glass
Traction Bronchiectasis	Micronodules

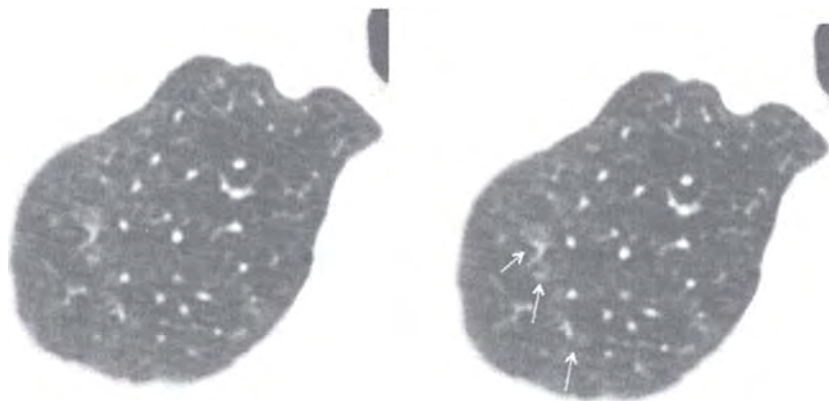
Trivia:

- * *NSIP is the most common Interstitial Lung Disease in Scleroderma*

RB-ILD and DIP : I* m going to discuss these two together because some people feel they are a spectrum. For sure they are **both smoking related diseases**.

- RB-ILD - Apical Centrilobular ground glass nodules
- DIP - More diffuse GGO, with patchy or subpleural distribution

RB-ILD: Respiratory Bronchiolitis + Symptoms = RB-ILD. This tends to be more upper lobe predominant (note that DIP tends to be more lower lobe predominant). Localized centrilobular ground glass nodules. The pathology tends to involve the entire cross section of lung.



RB-ILD - *Apical Centrilobular ground glass nodules + Smoking History*

DIP: Thought of as the end spectrum of RB-ILD. Peripheral lower lobe predominant ground glass, with small cystic spaces.

Sarcoid: This is a multi-system disease that creates “non-caseating granulomas.” The classic age is between 20-40. Along those lines, if the header to the question describes an African American female in her 20s-30s the answer is probably sarcoid. The lungs are by far the most common organ affected (90%).

Misc Trivia to know:

- Elevated angiotensin-converting enzyme (ACE)
- Hypercalcemia

Mediastinal lymph nodes are seen in 60-90% of times (classically in a 1 -2-3 pattern of bilateral hilars and right paratracheal). They have **perilymphatic nodules**, with an **upper lobe predominance**. Late changes include, upper lobe fibrosis, and traction bronchiectasis (honey combing is rare). Aspergillomas are common in the cavities of patient's with end stage sarcoid.

- * **1-2-3 Sign** - bilateral hila and right paratracheal
- * **Lambda Sign** - same as 1-2-3, but on Gallium Scan
- * **CT Galaxy Sign** - upper lobe masses (conglomerate of nodules) with satellite nodules

CXR can be used to “Stage” Sarcoid

Stage 0 = Normal

Stage 1 = Hilar / Mediastinal Nodes Only

Stage 2 = Nodes + Parenchyma Disease

Stage 3 = Parenchymal Disease

Stage 4 = End Stage (Fibrosis)

CHF - Congestive heart failure occurs because of cardiac failure, fluid overload, high resistance in the circulation, or some combination of the three. There are three phases of CHF, and these lend themselves to testable trivia.

Stages of CHF		
Stage 1 “ Redistribution”	Wedge Pressure 13-18	Cephalization of vessels, Big heart, Big Vascular Pedicle
Stage 2 “ Interstitial Edema”	Wedge Pressure 18-25	Kerley Lines, Peribronchial Cuffing, Less distinct contour of Central Vessels
Stage 3 “Alveolar Edema”	Wedge Pressure > 25	Airspace “fluffy” opacity, Pleural effusion

Right Heart Failure - This is less common than left heart failure, which ironically is the most common cause. Left heart failure causes pulmonary venous HTN which causes pulmonary artery HTN, which causes right heart failure. Some other less common causes of right heart failure include chronic PE, and right sided valve issues (tricuspid regurg). The signs of right heart failure include dilation of the azygos vein, dilation of the right atrium, dilation of the SVC, ascites, big liver, and contrast reflux into the hepatic veins on CTPA.

Lung Transplant Complications - Lung transplants are done for end-stage pulmonary disease (fibrosis, COPD, etc..)- The complications lend themselves easily to multiple choice test questions, and are therefore high yield. The best way to think about the complications is based on time.

Immediate Complications (< 24 hours)	
Donor-Recipient Size Mismatch	Mismatch up to 25% is ok. You can have a compressed lung (by the hyperexpanded emphysematous lung). Imaging is usually atelectasis.
Hyperacute Rejection	Secondary to HLA and ABO antigens. It's rapid and often fatal. Imaging shows massive homogenous infiltration
Early Complications (24 hours - 1 week)	
Reperfusion Injury	Peaks at day 4 as a non-cardiogenic edema related to ischemia-reperfusion. Typically improves by day 7.
Air Leak / Persistent Pneumothorax	Defined as a continuous leak for more than 7 days.
Intermediate Complication (8 days - 2 months)	
Acute Rejection	Ground Glass opacities and intralobular septal thickening. (No ground glass = no rejection). Improves with steroids.
Bronchial Anastomotic Complications	Leaks occur in the first month, stenosis can develop later (2-4 months).
Late Complications (2-4 months)	
CMV Infection	The most common opportunistic infection. Ground glass, tree-in-bud. Rare before 2 weeks.
Later Complications (> 4 months)	
Chronic Rejection	Bronchiolitis Obliterans; Affects 50% at 5 years. Bronchiectasis, bronchial wall thickening, air trapping.
Cryptogenic Organizing Pneumonia	Occurs with chronic rejection (but more commonly with acute rejection). Responds to steroids.
PTLD	Typically seen within the first year. EBV in 90%.
Upper Lobe Fibrosis	Associated with chronic rejection

Chronic Rejection / Bronchiolitis Obliterans Syndrome: This is the major late complication, that affects at least half of the transplants at 5 years (most commonly at 6 months). The term bronchiolitis obliterans is often used interchangeably with chronic rejection. The findings on CT include bronchiectasis, bronchial wall thickening, air trapping, and interlobular septal thickening. Just think air trapping on expiration seen at or after 6 months = chronic rejection.

Recurrence of Primary Disease after Transplant: For the purpose of multiple choice tests know that sarcoidosis is the most common recurrent primary disease (around 35%). Lots of other things can recur.

Lung Cancer after Transplant: Just remember that that native lung is still diseased, and can get cancer. The highest rate is with pulmonary fibrosis, and the most common risk factor is heavy tobacco use.

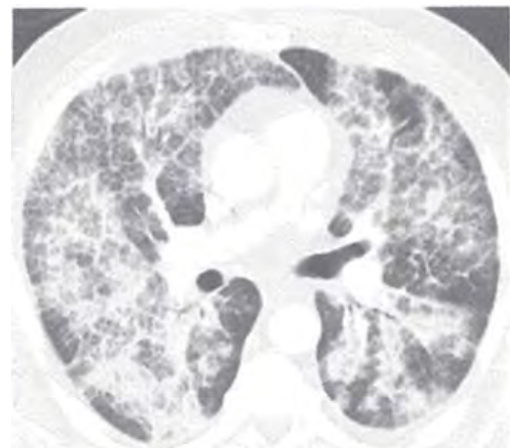
Alveolar Lung Disease:

Pulmonary Alveolar Proteinosis (PAP): They will show you a crazy paving lung (interlobular septal thickening, with ground glass). This can be primary (90%), or secondary (10%). Secondary causes include cancer or inhalation (silico-proteinosis).

Trivia Worth Knowing:

- * They are at increased risk of ***Nocardia infections***, and can be nocardia brain abscess.
- * ***Smoking is strongly associated with the disease.***
- * When seen in children (presenting before age 1) there is a known association with alymphoplasia.
- * Can progress to pulmonary fibrosis (30%).

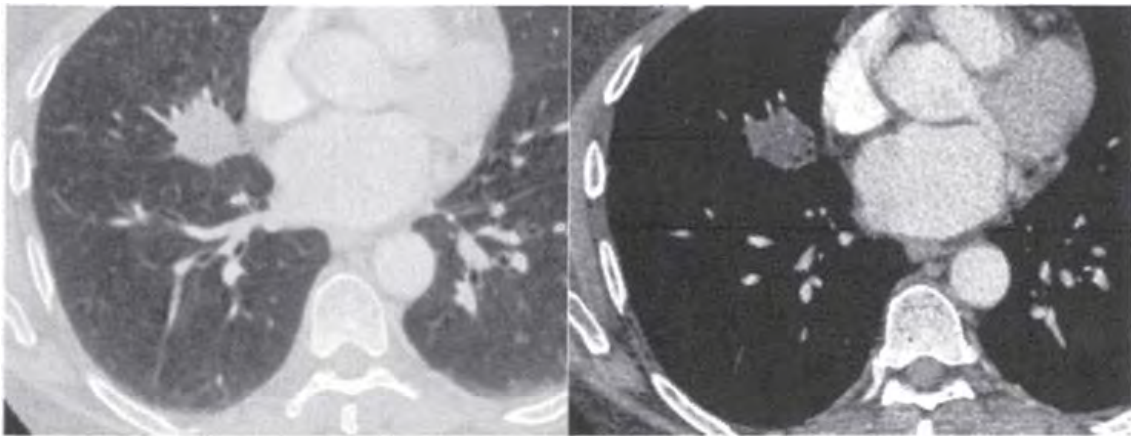
Crazy Paving - Interlobular septal thickening and ground glass. This isn't always PAP, in fact in real life that it is usually NOT PAP. There is a differential that includes common things like edema, hemorrhage, BAC, Acute Interstitial Pneumonia. Just know that for the purpose of multiple choice test the answer is almost always PAP.



Crazy Paving -ALWAYS PAP on Multiple Choice

Lipoid Pneumonia - There are actually two types; endogenous and exogenous.

- * **Exogenous:** This is seen in **old people who like to drink/aspirate mineral oil** (as a laxative). It can also be seen with the aspiration of vegetable oil or other animal oils. The look on plain film is an area of lung opacification that is chronic or slowly increases with time. The look on CT is a dead give away (and the most likely way this will be shown) with **low attenuation / fat density in the consolidation. Having said that this is also in the crazy paving differential.**
- * *Acute Exogenous Lipoid Pneumonia* - This is seen in children who accidentally poison themselves with hydrocarbons, or idiots trying to perform fire-eating or flame blowing.
- * **Endogenous** - This is actually more common than the exogenous type, and results from post obstructive processes (cancer) causing the building up of lipid laden macrophages.



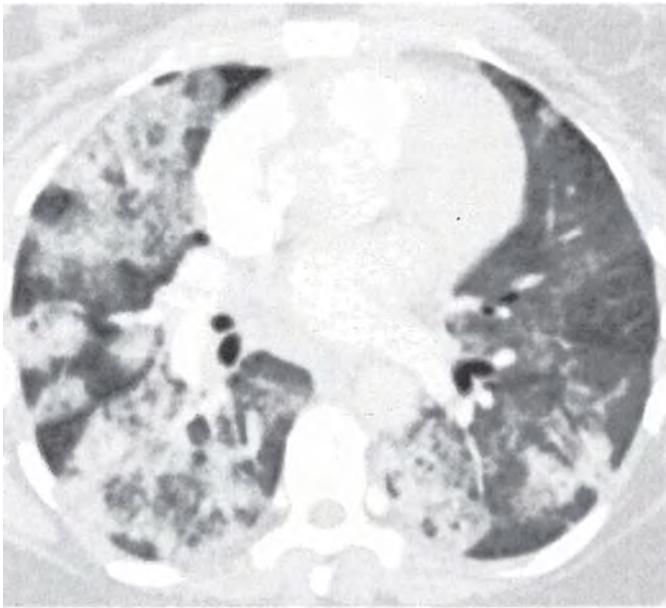
Lipoid Pneumonia – Fat Density in the consolidation

Organizing Pneumonia (*cryptogenic when cause not known "COP"*)

This used to be called BOOP, which was a lot more fun to say. There are lots of different causes; idiopathic, infection, drugs (amiodarone), collagen vascular disease, fumes etc... These guys respond well to steroids, and have an excellent prognosis.

Patchy air space consolidation or GGO (90%), in a **peripheral** or peri-bronchial distribution. Opacities tend to be irregular in shape. Findings of fibrosis are typically absent.

* **Reverse Halo (Atoll) Sign** is the classic Sign: Consolidation around a ground glass center



Cryptogenic Organizing Pneumonia -
-Reverse Halo Sign



Chronic Eosinophilic Pneumonia: Can be idiopathic or associated with a known antigen. Peripheral eosinophilia (blood test) is usually present. An asthma history is found in about 50% of cases. It looks exactly like COP. When you say COP you should say this one too (some people think it's the same disease as COP).

CT Findings: Peripheral GGO or consolidation. Upper lobes tend to be favored.

Hypersensitivity Pneumonitis: This is actually common. It's caused by inhaled organic antigens. It has acute, subacute, and chronic stages. Most of the time it's imaged in the subacute stage.

- Subacute: Patchy ground glass opacities. Ill-defined Centrilobular ground glass nodules (80%). Often has mosaic perfusion, and air trapping.
- * Chronic: Looks like UIP + Air trapping. You are gonna have traction bronchiectasis and air trapping. A **buzzword is "headcheese"** because it's a mix of everything (Ground Glass, Consolidation, Air-Trapping, and Normal Lung) **

General Ideas on Ground Glass vs Consolidative Opacity

Consolidation:

- * Most important question is Acute or Chronic:
 - *Acute:* Pneumonia, Edema, Hemorrhage, ARDS
 - *Chronic:* COP, Chronic Eosinophilic Pneumonia, BAC (adenocarcinoma in situ)

Ground Glass:

- * Most important question is Acute or Chronic:
 - *Acute:* Pulmonary Hemorrhage, Pulmonary Edema, Atypical Pneumonia (**PCP**, Viral), ARDS
 - *Chronic:* NSIP, Hypersensitivity pneumonitis, COP, Chronic Eosinophilic Pneumonia, BAC (adenocarcinoma in situ), Lipoid Pneumonia (zebra), PAP (rare in real life, common on tests).

Halo Signs

Reverse Halo (Atol)

-Central ground glass with rim of consolidation

DDx:

- COP (Classic)
- Fungal Pneumonia
- **TB**
- Wegeners
- Pulmonary Infarct

Halo

-Nodule with ground glass around it
-Represents hemorrhage / invasion into surrounding tissues

DDx:

- Invasive Aspergilosis (Classic)
- Other Fungus
- Hemorrhagic Mets
- Wegeners



Airways

Trachea: Has anterior horseshoes of cartilage. The transverse diameter should be no more than 2.5 cm (some are the transverse diameter of an adjacent vertebral body). There is a thin posterior membrane (that does NOT contain cartilage). This membrane can bow inward on expiratory CT (and this is normal).

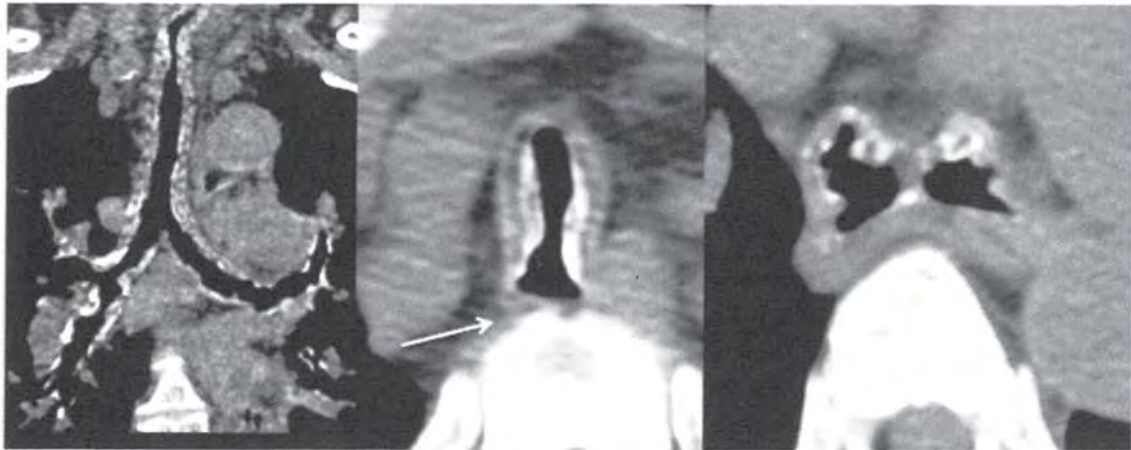
Tracheal Disease Game plan: Three big questions to ask yourself. (1) Is it focal or diffuse, (2) Does it involve the posterior membrane, and (3) is there calcification.

Relapsing Polychondritis: Spare the posterior membrane. Diffuse thickening of the trachea. No calcifications. Characterized by recurrent episodes of cartilage inflammation, and recurrent pneumonia.

Post Intubation Stenosis: Focal Subglottic **circumferential** stenosis, with an hourglass configuration.

Wegener's: **Circumferential** thickening, which can be **focal or long segment**. No calcifications. Subglottic involvement is common.

Tracheobronchopathia Osteochondroplastica (TBO): **Spare the posterior membrane**. You have development of **cartilaginous and osseous nodules** within the submucosa of the tracheal and bronchial walls.

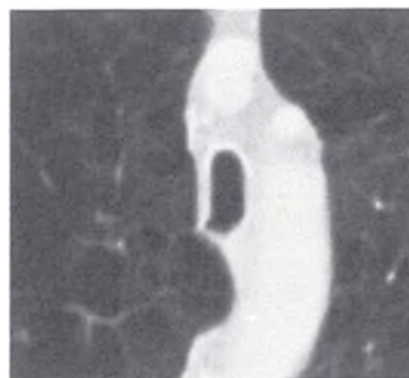


Tracheobronchopathia Osteochondroplastica (TBO) - Spares Posterior Membrane (arrow).

Amyloidosis: **Irregular** focal or short segment thickening, which can involve the posterior membrane. Calcifications are common.

Spares the Posterior Membrane		Does NOT Spare the Posterior Membrane	
Relapsing Polychondritis	Recurrent episodes of cartilage inflammation (ears, nose, joints, laryngeal and thyroid cartilage). Recurrent pneumonia is the most common cause of death.	Amyloid	Often confined to the trachea and main bronchi. Calcifications are common.
Tracheobronchopathia Osteochondroplastica (TBO):	Development of cartilaginous and osseous nodules. Typically occurs in men older than 50.	Wegeners	C-ANCA +, Sub-glottic trachea is the most common location.

Saber Sheath Trachea: Coronal diameter is less than two thirds of sagittal diameter. Affects older men with chronic obstructive pulmonary disease (COPD). The main bronchi will be normal in size. The tracheal wall will be normal in thickness.



Saber Sheath Trachea

Tracheal tumors - Tumors of the trachea are not common in the real world.

Tracheal Tumors	
Squamous Cell	Most Common. Associated with smoking, Often multifocal (10%), favors the lower trachea / proximal bronchus
Adenoid Cystic	2 nd Most common. Favors the upper trachea, and prefers the posterior lateral trachea. Has a variable look - can be a thickening, a mass, or a nodule.
Mets	Usually via direct extension (lung, thyroid, esophagus)
Squamous Cell Papilloma	Most common benign tumor. When it's a single papilloma think smoking. When it's multiple papillomas think HPV.

Cystic fibrosis - The sodium pump doesn't work and they end up with thick secretions and poor pulmonary clearance. The real damage is done by recurrent infections.

Things to know:

- *Bronchiectasis (begins as cylindrical and progresses to varicoid)*
- *It has an apical predominance (lower lobes are less affected)*
- *Hyperinflation*
- *Pulmonary Arterial Hypertension-*
- *Mucus plugging (finger in glove)*

Primary Ciliary Dyskinesia: Those little hairs in your lungs that clear secretions don't work. You end up with bilateral lower lobe bronchiectasis (remember that CF is mainly upper lobe). Other things these kids get is chronic sinusitis (prominent from an early age), and impaired fertility (sperm can't swim, girls get ectopics). They have chronic mastoid effusions, and conductive hearing loss is common. An important testable fact is that only **50% of the primary ciliary dyskinesia patients have Kartagener's Syndrome.**

Kartagener Syndrome: Primary Ciliary Dyskinesia + Situs Inversus.

CF	Primary Ciliary Dyskinesia
Abnormal Mucus, Cilia cannot move it	Normal Mucus, Cilia don't work
Normal Sperm, Absent Vas Deferens	Abnormal Sperm (they can't swim), Normal Vas Deferens
Upper lobe bronchiectasis	Lower lobe bronchiectasis



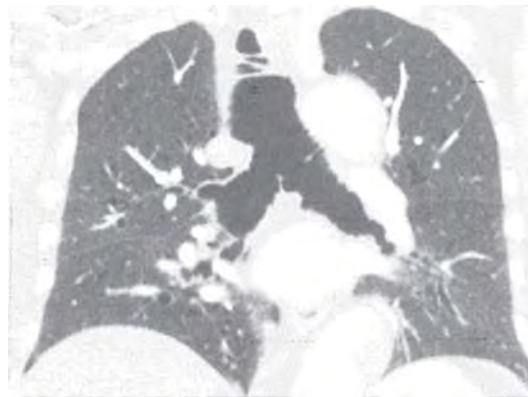
Williams Campbell

Syndrome - Huge zebra that is manifests as congenital cystic bronchiectasis from a deficiency of cartilage in the 4th-6th order bronchi.

✕

^ Mounier-Kuhn

(Tracheobronchomegaly) - There is a massive dilatation of the trachea (> 3cm). It's not well understood, and really the only thing that does this.



Mounier-Kuhn - Massively dilated trachea

Small Airways Disease

Bronchiolitis - This is an inflammation of the small airways. It can be infectious (like the viral patterns you see in kids) or inflammatory like RB-ILD in smokers, or asthma in kids.

Tree in Bud- This is a nonspecific finding that can make you think small airway disease. It's caused by dilation and impaction of the centrilobular airways. Because the centrilobular airways are centered 5-10mm from the pleural surface, that's where they will be. It's usually associated with centrilobular nodules.

Follicular Bronchiolitis - This is an inflammatory process seen in rheumatoid arthritis or Sjogrens. It's not well understood and related to some lymphoid hyperplasia. It looks like centrilobular ground glass nodules with scattered areas of bronchial dilation.

Constrictive Bronchiolitis - This is another inflammatory process that can be seen secondary to viral illness, transplant patients, drug reactions, or inhalation injury. It occurs secondary to mononuclear cells which form granulation tissue and plug the airway. You see air trapping on expiratory imaging. This is supposedly the cause of Swyer-Jame's hyperlucent lung.

Small Airway Disease	
Infectious Bronchiolitis	Tree-in-bud
RB-ILD	Smokers. Centrilobular ground glass nodules (upper lobe predominant)
Sub-acute Hypersensitivity Pneumonitis	Inhaling dust / other misc garbage. Centrilobular Ground glass nodules
Follicular Bronchiolitis	RA and Sjogrens. Centrilobular ground glass nodules
Constrictive Bronchiolitis	Viral, Drugs, Transplant, Inhalation. Air-Trapping.

Aspiration Pneumonia - Stroked-out old people and drunks love to aspirate. The testable trivia is to know the typical location of aspiration; posterior segment of upper lobes and superior segment of lower lobes if supine when aspirating, bilateral basal lower lobes in upright aspiration. May favor the right side, just like an ET tube. The most common complication is infection which can manifest as an empyema (which can get a bronchopleural fistula).

Aspiration Patterns (depends on what you aspirated)	
Aspiration of Gastric Acid "Mendelson's Syndrome"	Gives you an airspace opacity, if massive can look like pulmonary edema
Aspiration of water or neutralized gastric contents	"Fleeting Opacity" that resolves in hours
Aspiration of Bugs (often mouth bugs)	Gives you a real pneumonia, can get parapneumonic effusion, empyema, or even broncho-pleural fistula.
Aspiration of Oil (often mineral oil)	Lipoid Pneumonia. Will be low density

Pulmonary manifestations of systemic disease

Collagen vascular disease - Interstitial lung diseases are common in patients with collagen vascular diseases. I've tried to hit the high points of testable trivia.

Collagen Vascular Disease Pulmonary Manifestations		
Lupus	More pleural effusions and pericardiac effusions than with other connective tissue disease	Fibrosis is uncommon. Can get a "shrinking lung."
Rheumatoid Arthritis	Looks like UIP and COP. Lower lobes are favored.	Reticulations with or without honeycombing, and consolidative opacities which are organizing pneumonia
Scleroderma	NSIP > UIP; lower lobe predominant findings.	Look for the dilated fluid filled esophagus.
Sjogrens	LIP	Extensive ground glass attenuation with scattered thin walled cysts.
Ankylosing Spondylitis	Upper lobe fibrobullous disease	Usually unilateral first, then progresses to bilateral.

Caplan Syndrome = Rheumatoid Arthritis + Upper Lobe Predominant Lung Nodules. These nodules can cavitate, and there may also be a pleural effusion.

"Shrinking Lung" - This is a progressive loss of lung volume in both lungs seen in patients with **Lupus** ("Shrinking "L"ung for "SLe"). The etiology is either diaphragm dysfunction or pleuritic chest pain.

Hepatopulmonary syndrome - This is seen in liver patients with the classic history of *"shortness of breath when sitting up."* The opposite of what you think about with a CHF patient. The reason it happens is that they develop distal vascular dilation in the lung bases (subpleural telangiectasia), with dilated subpleural vessels that don't taper and instead extend to the pleural surface. When the dude sits up, these things engorge and shunt blood - making him/her short of breath. A Tc MAA scan will show shunting with tracer in the brain (outside the lungs). They have to either tell you the patient is cirrhotic, show you a cirrhotic liver, or give you that classic history if they want you to get this.

Wegener Granulomatosis - The classic triad is upper tract, lung, and kidneys (although this triad is actually rare). The lungs are actually the most common organ involved (95%). There is a highly variable look. The most common presentation is also probably the most likely to be tested; **nodules with cavitation**. The nodules tend to be random in distribution with about half of them cavitating. They can also show you ground glass changes which may represent hemorrhage.

Goodpasture Syndrome - Another autoimmune pulmonary renal syndrome. It favors young men. It's a super nonspecific look with bilateral coalescent airspace opacities that look a lot like edema (but are hemorrhage). They resolve quickly (within 2 weeks). If they are having recurrent bleeding episodes then they can get fibrosis. Pulmonary hemosiderosis can occur from recurrent episodes of bleeding as well, with iron deposition manifesting as small ill defined nodules.

Pleura, diaphragm and chest wall

Plaque - If they show you a pleural plaque they probably want you to say asbestos related disease. Remember the plaque doesn't show up for like 20-30 years after exposure. Remember that the pleural plaque of asbestosis typically spares the Costophrenic angles.

Pleural Calcifications (other than asbestos)

- * Old Hemothorax,
- * Old Infection,
 - TB,
 - Extraskelatal Osteosarcoma

Mesothelioma - The most common cancer of the pleura. About 80% of them have had asbestos exposure, and development is NOT dose dependent. The lag time is around 30-40 years from exposure. The **buzzword pleural rind** is worth knowing. The tendency is for direct invasion. Extension into the fissure is highly suggestive.

Fibrous tumors of the pleura - This is usually a solitary tumor arising from the visceral pleura. The key is to know that they are **NOT associated with asbestos**, smoking, or other environmental pollutants. They can get very large, and be a source of chest pain (although 50% are incidentally found). The second high yield testable fact is the association with **hypoglycemia and hypertrophic osteoarthropathy**.

Metastases - Here is the high yield trivia on this. As a general rule the subtype of adenocarcinoma is the most likely to met to the pleura. Lung cancer is the most common primary, with breast and lymphoma at 2nd and 3rd. Remember that a **pleural effusion is the most common manifestation** of mets to the pleura.

Lipoma - This is the most common benign soft tissue tumor of the pleura. The patients sometimes feel the “urge to cough.” They will not cause rib erosion. They “never” turn in a sarcoma. The differential consideration is extrapleural fat, but it is usually bilateral and symmetric.

Effusion - Some random factoids on pleural effusions that could be potentially testable. There has to be around 175cc of fluid to be seen on the frontal view (around 75cc can be seen on the lateral). Remember that medicine docs group these into transudate and exudate based on protein concentrations (Lights criteria). You are going to get elastic / compressive atelectasis of the adjacent lung.

Subpulmonic Effusion - A pleural effusion can accumulate between the lung base and the diaphragm. These are more common on the right, with “ski-slopping” or **lateralization of the diaphragmatic peak**. A lateral decubitus will sort it out in the real world.

Empyema - Basically this is an infected pleural effusion. It can occur with a simple pneumonia but is seen more in people with AIDS. Usually these are more asymmetric than a normal pleural effusion. Other features include; enhancement of the pleura, obvious septations, or gas.

Empyema vs Pulmonary Abscess	
Empyema	Pulmonary Abscess
Lentiform	Round
<i>Split Pleural Sign</i> (thickening and separation of the visceral and parietal pleura)	Claw Sign (acute angle with pleura)
Treated with chest tube	NOT treated with chest tube (risk of brochopleural fistula).

Empyema Necessitans - This is the fancy Latin word for when the empyema eats through the chest wall and into the soft tissues. It’s classically seen with **TB** (70%), with the second most common cause being actinomyces.

Diaphragmatic Hernia - These can be acquired via trauma, or congenital. The congenital ones are most common in the back left (Bochdalek), with anterior small and right being less common (Morgagni). The traumatic ones are also more common on the left (liver is a buffer).

Paralysis - This is a high yield topic because you can use fluoro to help make the diagnosis. Obviously the dinosaurs that write these tests love to ask about fluoro (since that was the only thing they did in residency). Diaphragmatic paralysis is actually idiopathic 70% of the time, although when you see it on multiple choice tests they want you to **think about phrenic nerve compression from a lung cancer**. Normally the right diaphragm is higher, so if you see an elevated left diaphragm this should be a consideration.

On a fluoroscopic sniff test you are looking for paradoxical movement (going up on inspiration - instead of down).

Mediastinal masses

Superior Mediastinal Masses: *These are often lumped into anterior mediastinal masses - as extension often occurs upward.*

* **“Superior Sulcus Tumor”** - Notice I didn’t call it a Pancoast tumor. To be a “Pancoast” tumor you need to have “Pancoast syndrome”, which is shoulder pain, C8-T2 radicular pain, and Horner Syndrome. The superior sulcus tumor can cause these but doesn’t have to. The most common tumor to do this is a squamous cell lung cancer (or bronchogenic adenocarcinoma - depending on what you read). Non-Small cell tumors may be a safer way to say it.

The Classic Question:

When is the superior sulcus (pancoast) tumor unresectable?

1. *Brachial Plexus involvement above T1 (C8 or higher)*
2. *Diaphragm Paralysis (infrs involvement of C3-4-5)*
3. *Greater than 50% vertebral body*
4. *Distal Nodes or Mets... etc..*

Anterior Mediastinal Masses:

- **Thymus:** The thymus can do a bunch of sneaky things. It can rebound from stress or chemotherapy and look huge. It can get cysts, cancer, carcinoid, etc...

Rebound vs Residual Lymphoma

- o **Rebound** - Discussed in detail in the Peds chapter. After stress or chemotherapy the thing can blow up 1.5 times the normal size and simulate a mass. Can be hot on PET.

- (1) PET might help - both are hot, but lymphoma is hotter.
- (2) MRI - Thymic Rebound should drop out on in-out of phase imaging (it has fat in it). Lymphoma will not drop out.

- o **Thymic Cyst** - Can be congenital or acquired. **Acquired is classic after thoracotomy, chemotherapy, or HIV.** They can be unilocular or multilocular. T2 bright is gonna seal the deal for you.
- o **Thymoma** - So this is kind of a spectrum ranging from non-invasive thyoma, to invasive thyoma, to thymic carcinoma. Calcification makes you think it's more aggressive. The thymic carcinomas tend to eat up the mediastinal fat and adjacent structures. The average age is around 50, and they are rare under 20. These guys can "drop met" into the pleural and retroperitoneum, so you have to image the abdomen.

High Yield Trivia: *Thymoma associations: Myasthenia Gravis, Pure Red Cell Aplasia, Hypogammaglobinemia.*

- o **Thymolipoma** - I only mention this zebra because it has a characteristic look. It's got a bunch of fat in it. Think "fatty mass with interspersed soft tissue." •

• Germ Cell Tumor - Almost always Teratoma (75%)

- o **Mediastinal Teratoma** - This is the most common extragonadal germ cell tumor. They occur in kids (below age 1) and adults (20s-30s). They are benign, but carry a small malignant transformation risk. Mature subtypes are equal in Men and Women, but immature subtypes are exclusively seen in men (which should be easy to remember). There is an **association with mature teratomas and Klinefelter Syndrome**. The imaging features include a **cystic appearance (90%), and fat**. They can have calcifications including teeth - which is a dead give away.

- * **Thyroid** - *Thyroid cancer and goiter are described in detail in the endocrine chapter.*
- * **Lymphoma** - *this is discussed in detail in the cancer section of this chapter.*
- * **Pericardial Cyst** - This is uncommon and benign. **The classic location is the right anterior cardiophrenic angle.** This classic location is the most likely question.

Middle Mediastinal Masses

- * **Fibrosing Mediastinitis** - This is a proliferation of fibrous tissue that occurs within the mediastinum. It's **classically caused by histoplasmosis** (but the most common cause is actually idiopathic). Other causes include TB, radiation, and Sarcoid. It's a **soft tissue mass with calcifications** that infiltrates the normal fat planes. It has been **known to cause superior vena cava syndrome.** It's associated with retroperitoneal fibrosis when idiopathic. *



Fibrosing Mediastinitis

- * **Bronchogenic Cyst** - These congenital lesions are usually within the mediastinum (most commonly found in the subcarinal space) or less commonly intraparenchymal. For the purpose of the CORE exam, they are going to be in the subcarinal region, causing obliteration of the azygoesophageal line on a CXR, and being waterish density on CT.
- * **Lymphadenopathy** - Could be mets, could be infection, could be reactive.
- * **Mediastinal Lipomatosis** - Excess unencapsulated fat seen in patients with iatrogenic steroid use, Cushings, and just plain old obesity.

Posterior Mediastinal Masses:

- Neurogenic - The most common posterior mediastinal mass is one of neurogenic origin. This includes schwannomas, neurofibromas, and malignant peripheral nerve sheath tumors.
- Bone Marrow - Extramedullary hematopoiesis (EMH) is a response to failure of the bone marrow to respond to EPO. Classic conditions include CML, Polycythemia vera, myelofibrosis, sickle cell, and thalassemia.

Pulmonary arteries

Pulmonary Embolism: This is a significant cause of mortality in hospitalized patients. The gold standard is catheter angiography, although this is invasive and carries risks. As a result tests like the D-Dimer (which has an almost 100% negative predictive value), and the DVT lower extremity ultrasound were developed. Now, the CTPA as the primary tool.

Historical “High-Yield” Signs of PE on a CXR	
Westermarck Sign	Regional Oligemia
Fleishner Sign	Enlarged Pulmonary Artery
Hampton’s Hump	Peripheral Wedge Shaped opacity
Pleural Effusion	Obviously not specific, but seen in 30% of PEs.

Differentiating acute vs chronic PE is a high yield tool.

Acute vs Chronic PE	
Acute	Chronic
Central	Peripheral
Venous Dilation	Shrunken Veins with collateral vessels
Perivenous soft tissue edema	Calcifications within the thrombi, and within the venous walls

Pulmonary infarct mimics - A pulmonary infarct is a wedge shaped opacity, that is going to “melt” (resolve slowly), and sometimes can cavitate. Obviously a cavitary lesion throws up lots of flags and makes people say TB, or cancer. When it’s an opacity in the lung and the patient doesn’t have a fever sometimes people think cancer - plenty of pulmonary infarcts have been biopsied.

Pulmonary Artery Aneurysm / Pseudoaneurysm.- Think about three things for multiple choice; (1) **Iatrogenic from swan ganz catheter *most common** (2) Behcets, (3) Chronic PE. When they want to lead swan ganz they may say something like “patient in the ICU.” The buzzwords for Behcets are: “Turkish descent”, and “mouth and genital ulcers.”

- * **Hughes-Stovin Syndrome:** This is a zebra cause of pulmonary artery aneurysm that is similar (and maybe the same thing) as Behcets. It is characterized by recurrent thrombophlebitis and pulmonary artery aneurysm formation and rupture.
- * **Rasmussen Aneurysm:** This has a cool name, which instantly makes it high yield for testing. This is a pulmonary artery **pseudoaneurysm secondary to pulmonary TB**. It usually involves the upper lobes in the setting of reactivation TB.
- * **Tetralogy of Fallot Repair Gone South:** So another possible testable scenario is the patch aneurysm, from the RVOT repair.

Pulmonary Hypertension - Pulmonary arterial pressures over 25 are going to meet the diagnosis. I prefer to use the “outdated” primary and secondary way of thinking about this.

Primary: The idiopathic type is very very uncommon, seen in a small group of young women in their 20s.

Secondary: This is by far the majority, and there are a few causes you need to know: Chronic PE , Right Heart Failure/ Strain, Lung Parenchymal Problems- (This would include emphysema, and various causes of fibrosis). COPDers with a pulmonary artery bigger than the aorta (A/PA ratio) have increased mortality (says the NEJM).

Imaging Signs of Pulmonary HTN: The big pulmonary artery (> 29mm), or larger than the aorta. Mural calcifications of central pulmonary arteries (seen in Eisenmenger phenomenon). Right ventricular dilation and hypertrophy. Centrilobular ground-glass nodules.

Pulmonary Veno-Occlusive Disease: Uncommon variant of primary pulmonary hypertension, that affects the post capillary pulmonary vasculature. For **gamesmanship PAH + Normal Wedge**, you should think this. The normal wedge pressure differentiates it from other post capillary' causes; such as left atrial myxoma, mitral stenosis, and pulmonary vein stenosis

Trauma:

Diaphragmatic Injury - There is a lot of testable trivia regarding diaphragmatic injury and therefore it is probably the most high yield subject with regard to trauma:

Things to know:

- * Left side is involved 3 times more than the right (liver is a buffer)
- * Most ruptures are “radial”, longer than 10cm, and occur in the posterior lateral portion
- * **Collar Sign** - This is sometimes called the hour glass sign, is a waist-like appearance of the herniated organ through the injured diaphragm
- * **Dependent Viscera Sign** - This is an absence of interposition of the lungs between the chest wall and upper abdominal organs (liver on right, stomach on left).

Tracheo-Bronchial Injury: Airway injury is actually pretty uncommon. When it does occur it's usually within 2cm of the carina. **Injury close to the carina is going to cause a pneumomediastinum rather than a pneumothorax** - that is a testable fact. When you get a tracheal laceration, it most commonly occurs at the junction of the cartilaginous and membranous portions of the trachea.

Macklin Effect: This is probably the most common cause of pneumomediastinum in trauma patients (and most people haven't heard of it). The idea is that you get alveolar rupture from blunt trauma, and the air dissects along bronchovascular sheaths into the mediastinum.

Boerhaave Syndrome: You probably remember this from step 1. The physical exam buzzword was “Hammonds Crunch.” Basically you have a ruptured esophageal wall from vomiting, resulting in pneumomediastinum / mediastinitis.

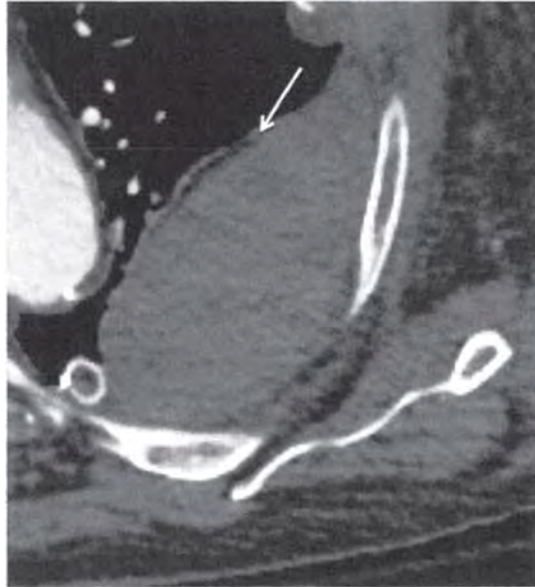
Flail Chest: This is 3 or more segmental (more than one fracture in a rib) fractures, or more than 5 adjacent rib fractures. The physical exam buzzword is “paradoxical motion with breathing.”

Pneumothorax: Obviously you don't want to miss the tension pneumothorax. The thing they could ask is “inversion or flattening of the ipsilateral diaphragm.”

Malpositioned Chest Tubes: Sometimes the ED will ram them into the parenchyma. This is more likely to occur in the setting of background lung disease or pleural adhesions. You'll see blood around the tube. Bronchopleural fistula may occur as a sequela. The placement of a tube in a fissure is sorta controversially bad (might be ok).

Hemothorax: If you see pleural fluid in the setting of trauma, it's probably blood. The only way I can see them asking this is a density question; a good density would be **35-70 H.U.**

Extrapleural Hematoma: This is a little tricky, and they could show you a picture of it. If you have an injury to the chest wall that damages the parietal pleura then you get a hemothorax. If you have an injury to the chest wall, but your parietal pleura is still intact you get an extrapleural hematoma. The **classic history is “persistent fluid collection after pleural drain/tube placement.”** The **buzzword / sign is displaced extrapleural fat.** There is a paper out there that suggests a biconvex appearance is more likely arterial and should be watched for rapid expansion. This may be practically useful, but is unlikely to be asked. Just know the classic history, and displaced extrapleural fat sign.



Extrapleural Hematoma
Displaced Extrapleural Fat (arrow)

Pulmonary Contusion: This is the most common lung injury from blunt trauma. Basically you are dealing with alveolar hemorrhage without alveolar disruption. The typical look is non-segmental ill defined areas of consolidation with **sub pleural sparing**. Contusion should appear within 6 hours, and disappear within 72 hours (if it lasts longer it's probably aspiration, pneumonia, or a laceration).

Pulmonary Laceration: So a tear in the lung will end up looking like a pneumatocele. If they show you one it will probably have a **gas -fluid (blood) level** in it. These things can be masked by surrounding hemorrhage early on. The major difference between contusion and laceration is that a laceration **resolves much more slowly** and can even produce a nodule or a mass that persists for months.

Aorta - The aorta is injured **most commonly at the aortic isthmus** (some sources say 90%). The second and third most common locations are the root and at the diaphragm. Some people say the root is actually the most common, but most of these people die prior to making it to the hospital. This is a minority opinion. If asked what is the most common site of traumatic aortic injury the answer is isthmus. It's usually obvious on a candy cane CTA. The main mimic would be a “ductus bump” which is a normal variant. The way to tell (if it isn't obvious) is the presence of secondary signs of trauma (mediastinal hematoma).

Blunt Cardiac Injury: If you have hemopericardium in the setting of trauma, you can suggest this and have the ED correlate with cardiac enzymes and EKG findings.

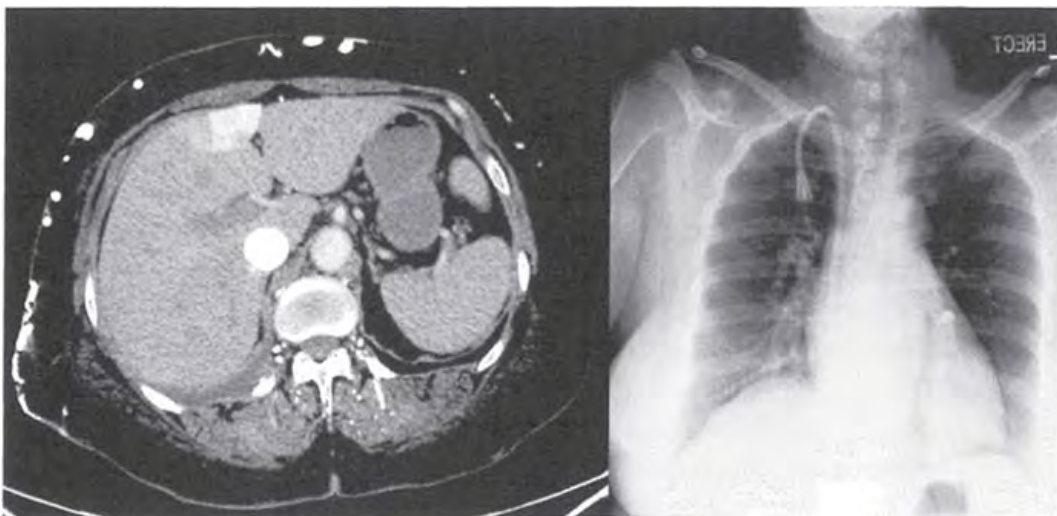
Fat Embolization Syndrome: This is seen in the setting of a long bone fracture or Intramedullary rod placement. You get fat embolized to the lungs, brain and skin (clinical triad of rash, altered mental status, and shortness of breath). The timing is **1-2 days after the femur fracture**. The lungs will have a ground glass appearance that makes you think **pulmonary edema**. You will not see a filling defect - like a conventional PE. If they don't die, it gets better in 1-3 weeks.

Barotrauma: Positive pressure ventilation can cause alveolar injury, with air dissecting into the mediastinum (causing pneumomediastinum, and pneumothorax). Patients with acute lung injury or COPD have a high risk of barotrauma from positive pressure ventilation. Lungs with pulmonary fibrosis are actually protected because they don't stretch.

Lines / Devices:

Central Lines: The main way to ask questions about central lines is to show them being malpositioned and asking you where they are. An abrupt bend at the tip of the catheter near the cavo-atrial junction should make you think azygos. If it's on the left side of the heart, it's either (1) arterial or (b) in a duplicated SVC.

This is a sneaky trick, related to Central Lines. They can show you the pseudo lesion / hot quadrate sign (seen with SVC syndrome), and then show you a CXR with a central venous catheter. The idea is that **central lines are a risk factor for SVC occlusion**.



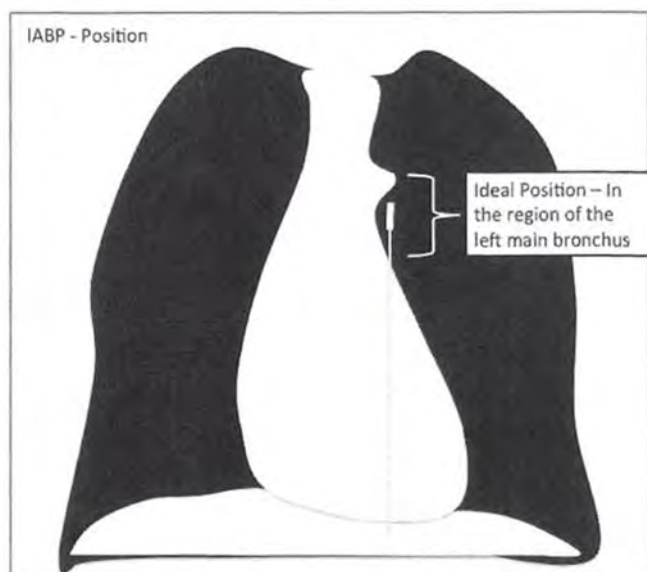
Hot Quadrate Sign from - SVC Obstruction

Endotracheal Tube (ETT) Positioning - The tip of the ETT should be about 5cm from the carina (halfway between the clavicles and the carina). The tip will go down with the chin tucked, and up with the chin up (“the hose goes, where the nose goes”). Intubation of the right main stem is the most common goof (because of the more shallow angle) - this can lead to left lung collapse. You can sometimes purposefully intubate one lung if you have massive pulmonary hemorrhage (lung biopsy gone bad), to protect the good lung.

Intra-Aortic Balloon Pump (IABP) - This is used in cardiogenic shock to help with “diastolic augmentation,” - essentially providing some back pressure so the vessels of the great arch (including the coronaries) enjoy improved perfusion.

For the purpose of multiple choice tests you can ask three things:

- (1) *What is the function?* - decrease LV afterload, and increased myocardial perfusion,
- (2) *What is the correct location?* the balloon should be located in the proximal descending aorta, just below the origin of the left subclavian artery (balloon terminates just above the splanchnic vessels),
- (3) *Complications?* - dissection during insertion, obstruction of the left subclavian from malpositioning.



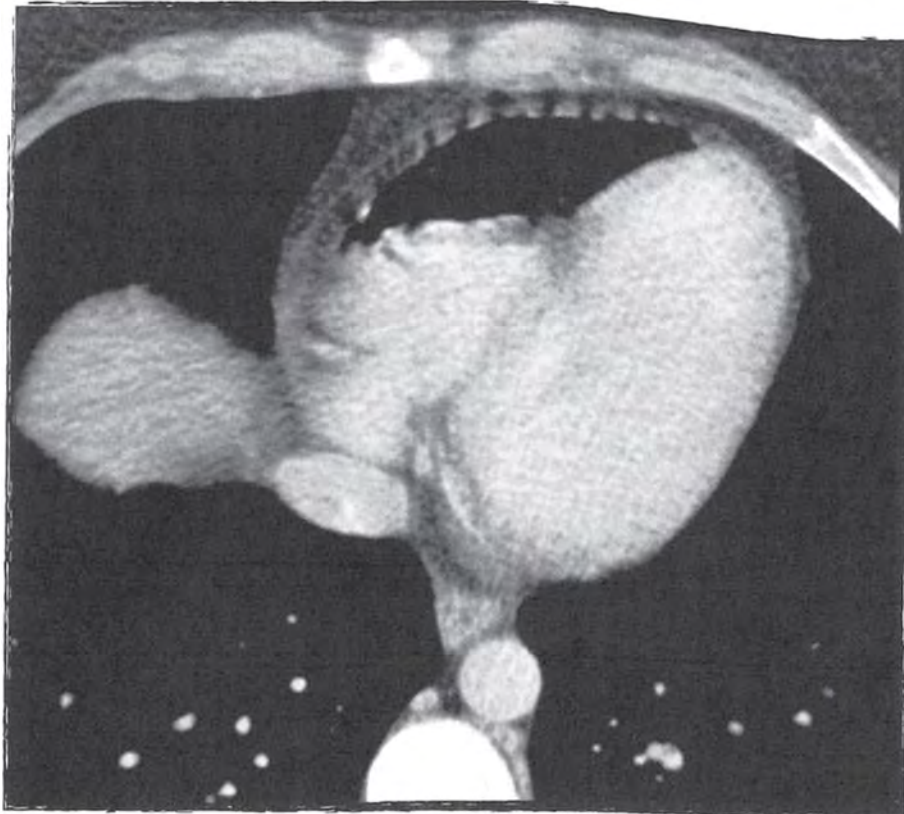
ACR Appropriateness Criteria: This is high yield trivia.

- * First Line for Suspected Metastatic Disease = CXR
- * Recommendation for patients on mechanical ventilation = Daily CXR
- * First Line for Chest Pain and High Suspicion for Aortic Dissection = CXR

7

Cardiac

Prometheus Lionhart, M.D.



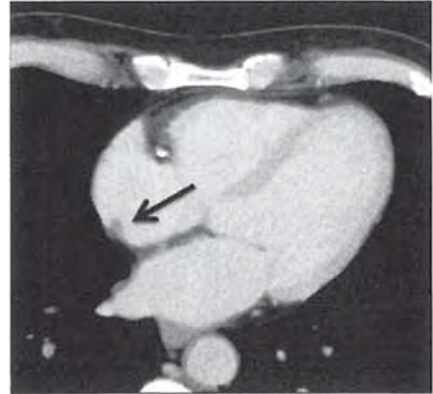
Cardiac imaging is universally dominated in every sense of the word by Cardiologists. Having said that, you are still responsible for cardiac imaging. Interestingly, up until about 25 years ago, Radiology Residents were tested on cardiac cath knowledge on oral boards until the ABR finally admitted that battle was lost.

High Yield Topics:

- Congenital Heart Disease
- Cardiac MRI

Chambers

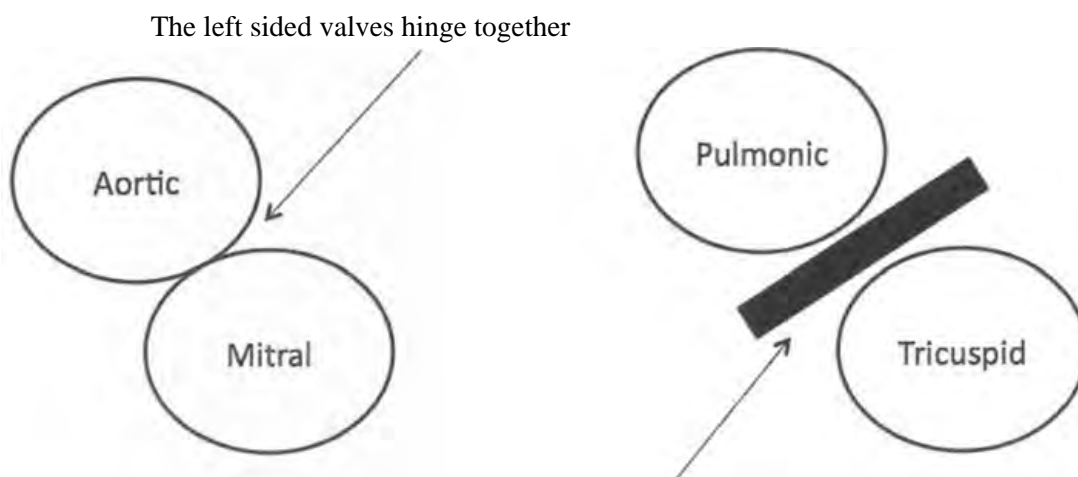
Right Atrium: Defined by the IVC. The **Crista Terminalis** is a frequently tested normal structure (it's not a clot or a tumor). It is a muscular ridge that runs from the entrance of the superior- to that of the inferior vena cava. Another normal anatomic structure that is frequently shown (usually on IVC gram) is the IVC valve or **Eustachian valve**. It looks like a little flap in the IVC as it hooks up to the atrium. When the tissue of this valve has a more trabeculated appearance it is called a **Chiari Network**.



Crista Terminalis (its not a tumor, or a clot)

Coronary Sinus: The main draining vein of the myocardium. It runs in the AV groove on the posterior surface of the heart and enters the right atrium near the tricuspid valve

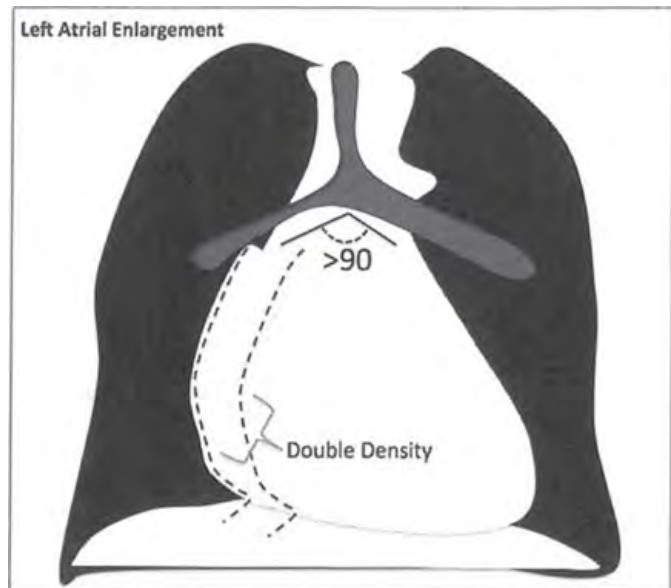
Right Ventricle: Defined by the Moderator Band. Has several characteristics that are useful for distinguishing it (and make good test questions). The tricuspid **papillary muscles insert on the septum** (not the case with the mitral valve). There is no fibrous connection between the AV valve / outflow tract. The pulmonary valve has three cusps, and is separated from the tricuspid valve by a thick muscle known as the crista supraventricularis . This differs from the left ventricular outflow tract, where the mitral and aortic valves lie side by side.



The right sided valves have an infundibulum between them

Left Atrium: The most posterior chamber. When you think about multiple choice questions regarding the left atrium, think about the various signs of enlargement.

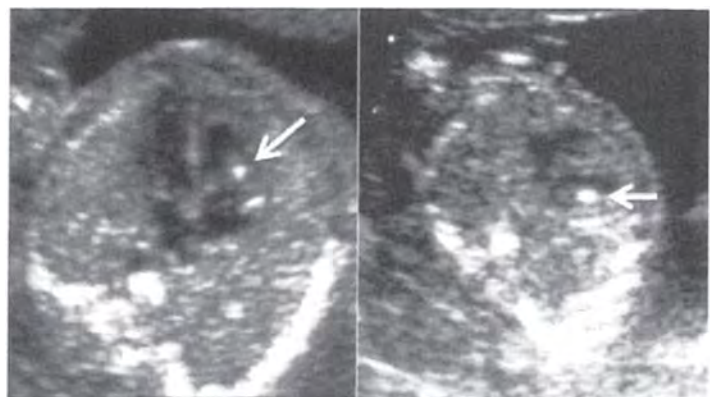
- * *Double Density (direct sign): Superimposed second contour on the right heart, from enlargement of the right side of the left atrium*
- * *Splaying of the Carina (indirect sign): Angle over 90 degrees suggests enlargement*
- * *Walking Man Sign (indirect sign): Posterior displacement of the left main stem bronchus on lateral radiograph. This creates an upside down "V" shape with the intersection of the right bronchus (looks like a man walking).*



Left Ventricle: The leaflets of the mitral valve are connected to the papillary muscles via cord-like tendons called chordae tendinae. The papillary muscles insert into the lateral and posterior walls as well as the apex of the left ventricle (not the septum, as is the case on the right).

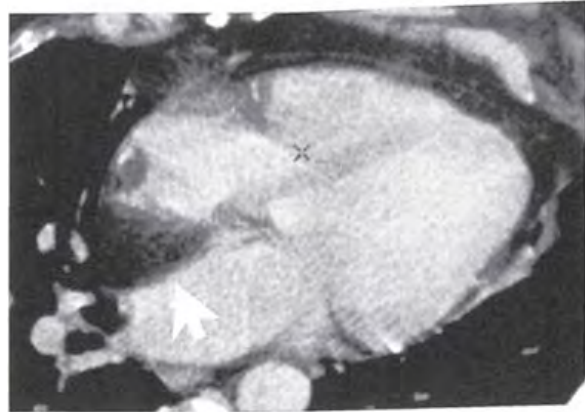
Echogenic Focus in Left

Ventricle: Relatively common sonographic observation seen on pre-natal ultrasound. It is a calcified papillary muscle that usually goes away by the third trimester. So who gives a shit? Well they are **associated with an increased incidence of Downs** (13%). Don't get it twisted, having one means nothing other than you should look for other signs of downs (most of the time it's normal).



Echogenic Focus in Left Ventricle

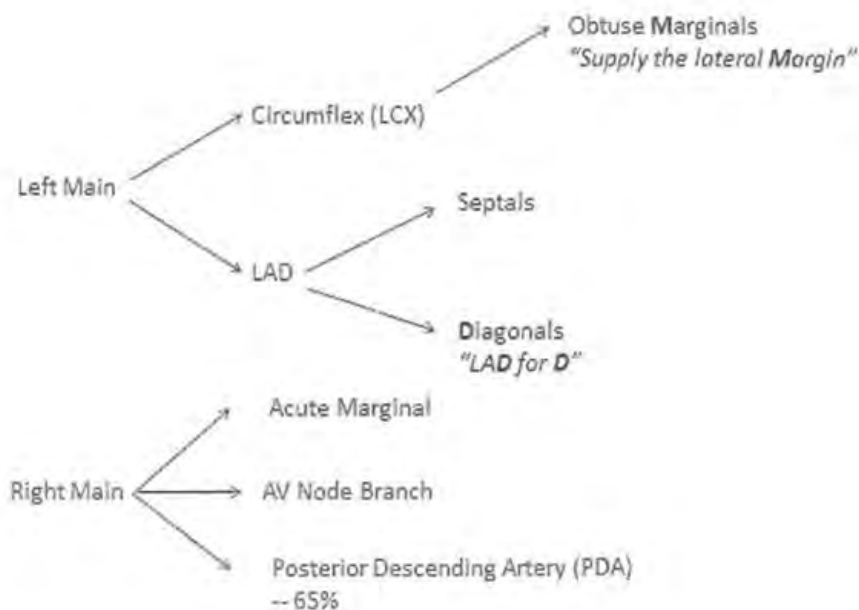
Lipomatous Hypertrophy of the Intra-Atrial Septum: This has a very classic look of a dumbbell (bilobed) appearance of fat density in the atrial septum, sparing the fossa ovalis. It **sparing the fossa ovalis**, creating a dumbbell appearance (*when it doesn't spare it think lipoma*). It's associated with being fat and old. As a point of trivia it can cause supraventricular arrhythmia, although usually does nothing. Additional even more high-yield trivia is that it **can be hot on PET because it's often made of brown fat**.



Coronaries

Questions regarding the coronaries will likely come in two flavors: Normal (which will be mostly vocab), and Abnormal (which will only have one or two pathologies).

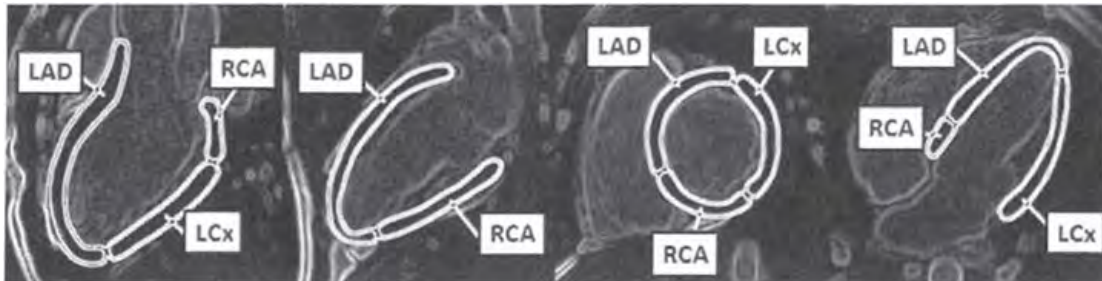
Normal: There are three coronary cusps; right, left, and non-coronary (posterior). The left main comes off the left cusp, the right main comes off the right cusp.



With regard to what perfuses what, the following are high yield factoids:

- * RCA perfuses SA node 60%
- * RCA perfuses AV node 90%

Typical vascular perfusion territories are a high yield topic.

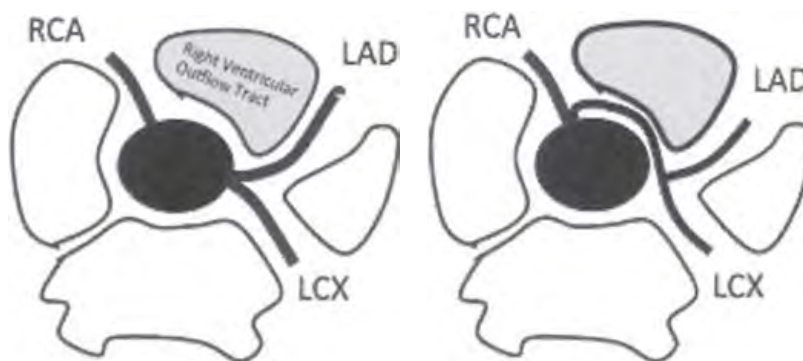


Dominance: Coronary Dominance is determined by **what vessel gives rise to the posterior descending artery and posterior left ventricular branches** (most are **right - 85%**). You can be “co-dominant” if the posterior descending artery arises from the right coronary artery and the posterior left ventricular branches arise from the left circumflex coronary artery

Not Normal: **Anomalies of the Origin, Course, and Termination:**

Malignant Origin: Most Common and Most Serious: **LCA from the Right Coronary Sinus**, coursing between the Aorta and Pulmonary Artery. This guy can get compressed and cause sudden cardiac death.

- * Anomalous right off the left cusp — Repair if symptomatic
- * Anomalous left of the right cusp — Always Repair



Normal Anatomy interarterial LCA
(the bad one)

ALCAPA: Anomalous Left Coronary from the Pulmonary Artery. There are two types: (a) Infantile type (they die early), and (b) adult (still at risk of sudden death). The multiple choice question is going to be “**STEAL SYNDROME**” - which describes a reversal of flow in the LCA as pressure decreases in the pulmonary circulation.

Myocardial Bridging: This is an intramyocardial course of a coronary artery (usually the LAD). The finding may cause symptoms as the diameter decreases with systole, or may cause an issue for CABG planning. This can be a source of ischemia.

Coronary Artery Aneurysm: By definition this is a vessel with a diameter greater than 1,5x the normal lumen. Most common cause is atherosclerosis. Most common cause in child is Kawasaki (spontaneously resolves in 50%). They can occur from lots of other vasculitis as well. Last important cause is iatrogenic (cath).

Coronary Fistula: Defined as a connection between a coronary artery and cardiac chamber or great vessels. It's usually the RCA, with drainage into the right cardiac chambers. They are associated / result in coronary aneurysm. *If you see big crazy dilation of the coronaries - think about this.*

Coronary CT

Who is the ideal patient to get a coronary CT? There are two main groups of people getting these. (1) Low risk or atypical chest pain patients. A negative coronary CT will help stop a stress test or cath from occurring. Why do a procedure with risks on someone with GERD? (2) Suspected aberrant coronary anatomy.

What is the ideal heart rate? To reduce motion related artifacts a slow heart rate is preferred. Most books will tell you under 60 beats per min. Beta blockers are used to lower the heart rate to achieve this ideal rate.

Are there contraindications to beta blockers? Yup. Patients with severe asthma, heart block, acute chest pain, or recent snorting of cocaine - should not be given a beta blocker.

What if I can't give the beta blocker? Can he still have the scan? Yes, you just can't use a prospective gating technique. You'll have to use retrospective gating.

What is the difference between prospective and retrospective gating?

-Prospective: "Step and Shoot" - R-R interval

- Pro: There is reduced radiation b/c the scanner isn't on the whole time
- Con: No functional imaging
- Trivia: Always axial, not helical

-Retrospective: Scans the whole time, then back calculates

- Pro: Can do functional imaging
- Con: Higher radiation (use of low pitch - increases dose)
- Trivia: this is helical

Other than beta blockers, are any other drugs given for coronary CT? Yup.

Nitroglycerine is given to dilate the coronaries (so you can see them better).

Are there contraindications to nitroglycerine? Yup. Hypotension (SBP < 100), severe aortic stenosis, hypertrophic obstructive cardiomyopathy, and Phosphodiesterase (Viagra-Sildenafil) use.

Valvular

Velocity-encoded cine MR imaging (VENC), also known as velocity mapping or phase-contrast imaging, is a technique for quantifying the velocity of flowing blood.

Aortic Stenosis: This may be congenital (bicuspid) or Acquired (Degenerative or Rheumatic Heart). Increased afterload can lead to concentric LV hypertrophy. Peak velocity through the valve can be used to grade the severity. Velocity-encoded cine MR imaging (VENC), which also answers to the name “velocity mapping” or “phase-contrast imaging”, is an MRI technique for quantifying the velocity of flowing blood (if anyone would happen to ask).

Dilation of ascending aorta is due to jet phenomenon related to a stenotic valve. Comes in three flavors: (a) valvular, (b) subvalvular, (c) and supravalvular. Valvular is the most common (90%).

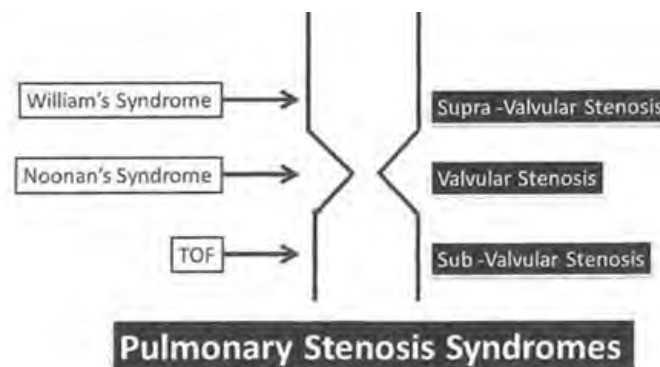
- * **When I say “Supra-valvular Aortic Stenosis” you say Williams Syndrome**
- **When I say “Bicuspid Aortic Valve and Coarctation” you say Turners Syndrome**

Aortic Regurgitation: Seen with bicuspid aortic valves, bacterial endocarditis, Marfan’s, aortic root dilation from HTN, and aortic dissection. How rapid the regurgitation onsets determines the hemodynamic impact (acute onset doesn’t allow for adaptation).

Mitral Stenosis: Rheumatic heart disease is the most common cause. The case could be shown as a CXR with left atrial enlargement (double density sign, splaying of the carina, posterior esophageal displacement).

Mitral Regurgitation: The most common acute causes are endocarditis or papillary muscle / chordal rupture post MI (Step 1 question was “Austin Flint Murmur”). The chronic causes can be primary (myxomatous degeneration) or secondary (dilated cardiomyopathy leading to mitral annular dilation). Remember the isolated Right Upper Lobe pulmonary edema is associated with mitral regurgitation.

Pulmonary Stenosis: Just like in the Aortic Valve, comes in three flavors: (a) valvular, (b) subvalvular, (c) and supravalvular. Valvular is the most common, and can lead to ventricular hypertrophy. Associated with **Noonan Syndrome** (male version of turners). “**Peripheral Pulmonary Stenosis**” is seen with **Alagille syndrome** (kids with absent bile ducts). Williams can give you supra-valvular aortic stenosis (and pulmonic).



Pulmonary Regurgitation: The most common situation for this is congenital valve disease after valve surgery. The classic scenario is actually TOF patient who has been repaired.

Tricuspid Regurgitation: Most common form of tricuspid disease, due to the relatively weak annulus (compared to the mitral). May occur in the setting of **endocarditis (IV drug use)**, or **carcinoid syndrome** (serotonin degrades the valve). **The most common cause in adults is pulmonary arterial hypertension.**

Ebstein Anomaly: Seen in children whose moms used Lithium (most cases are actually sporadic). The tricuspid valve is hypoplastic and the posterior leaf is displaced apically (downward). The result is enlarged RA, decreased RV (“atrialized”), and tricuspid regurgitation. They have the massive “box shaped” heart on CXR.

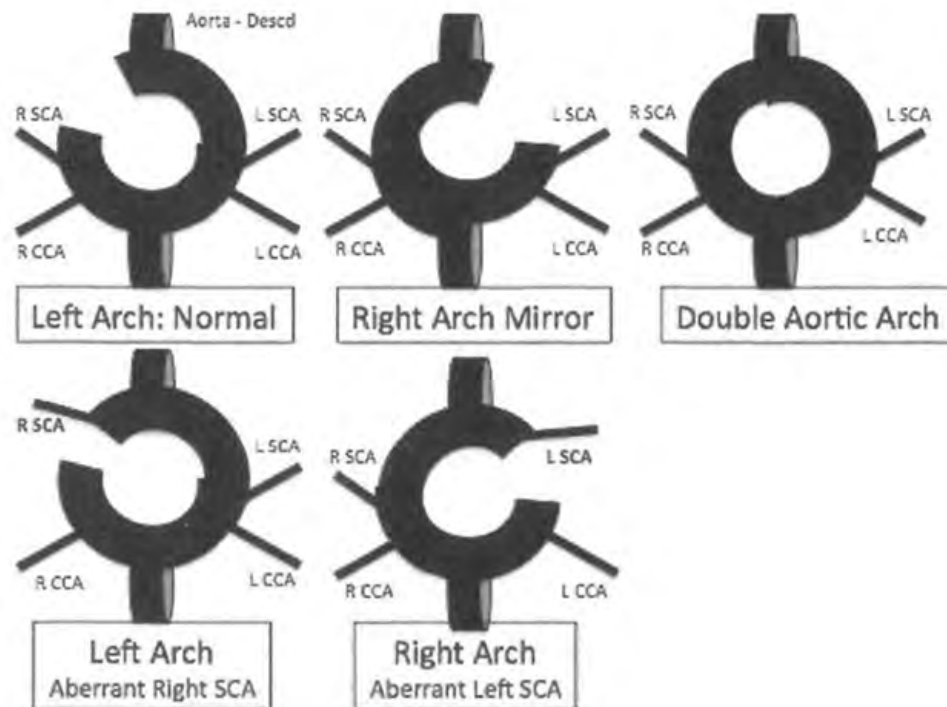
Tricuspid Atresia: Congenital anomaly that occurs with RV hypoplasia. Almost always has an ASD or PFO. Recognized association with asplenia. Can have a right arch (although you should think Truncus and TOF first). As a point of confusing trivia; tricuspid atresia usually has pulmonary stenosis and therefore will have decreased vascularity. If no PS is present, there will be increased vascularity.

Carcinoid Syndrome: This can result in valvular disease, but only after the tumor has met’d to the liver. The serotonin actually degrades heart valves, **typically both the tricuspid and pulmonic valves**. Left sided valvular disease is super rare since the lungs degrade the vasoactive substances. When you see right sided disease you should think of two scenarios: (1) primary bronchial carcinoid, or (2) right-to-left shunts.

Great Vessels

The most common variant in branching is the “bovine arch” in which the brachiocephalic artery and right common carotid artery arise from a common origin. The terminology right arch / left arch is described based on the aortic arch’s relationship to the trachea. There are 5 types of right arches, but only two are worth knowing (Aberrant Left, and Mirror Branching).

When I say Right Arch with Mirror Branching, You say congenital heart.



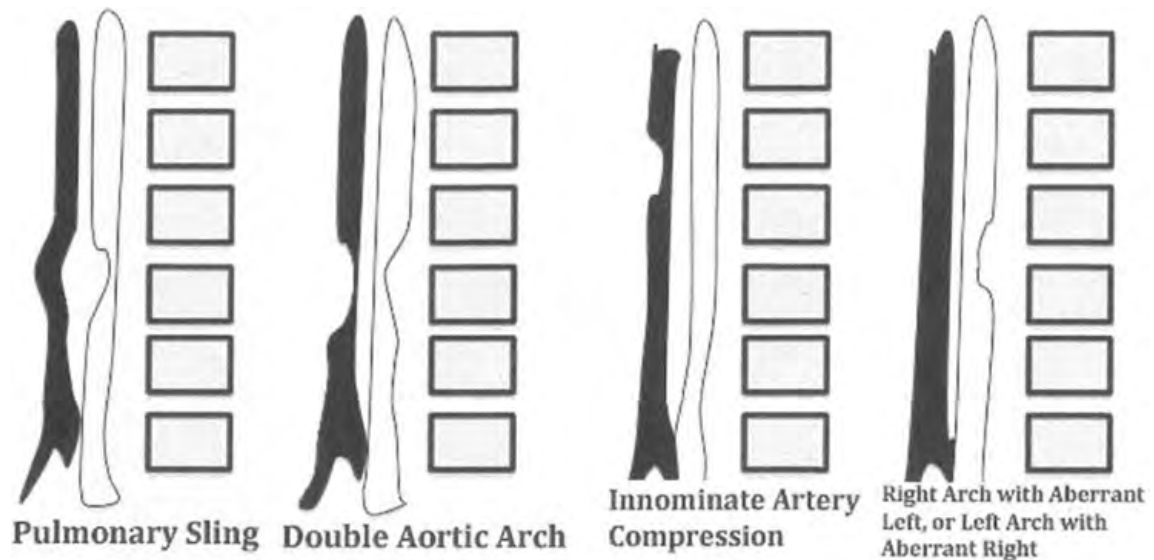
Right Arch with Aberrant Left Subclavian: The last branch is the left aberrant subclavian artery.

Right Arch with Mirror Branching: Although these are often asymptomatic they are strongly associated with congenital heart disease. Most commonly they are associated with TOF. However, they are most closely associated with Truncus. Obviously, this tricky wording lends itself nicely to a trick question.

- * *If there is a mirror image right arch, then 90% will have TOF (6% Truncus).*
- * *If the person has Truncus, then they have a mirror image right arch 33% (TOF 25%).*

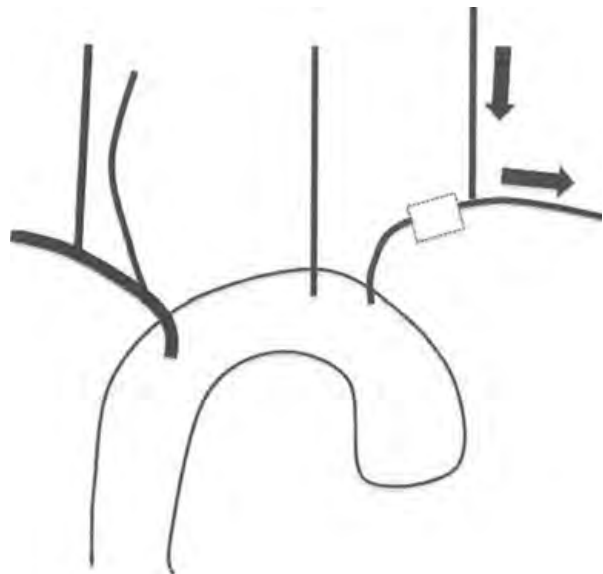
Left Arch Aberrant Right Subclavian: The most common arch anomaly. Although it is usually asymptomatic it can **sometimes be associated with dysphagia lusoria**, as the RSCA passes posterior to the esophagus. The last branch is the right aberrant subclavian artery. The **origin of the RSCA may be dilated = Diverticulum of Kommerell**.

Double Aortic Arch: *The most common vascular ring.* As a point of trivia, symptoms may begin at birth and include tracheal compression and/or difficulty swallowing. The right arch is larger and higher, and the left arch is smaller and lower. Arches are posterior to esophagus and anterior to trachea.



Subclavian Steal Syndrome/Phenomenon: So there is a “Syndrome” and there is a “Phenomenon.”

- * *SS Phenomenon:* Stenosis and/or occlusion of the proximal subclavian with retrograde flow in the ipsilateral vertebral artery. *
- * *SS Syndrome:* Stenosis and/or occlusion of the proximal subclavian artery with retrograde flow in the ipsilateral vertebral artery AND associated cerebral ischemic symptoms.



Subclavian Steal: Occlusion proximal to the vertebral in the SVC can result in retrograde flow from the vertebral

If the level of stenosis and/or occlusion is proximal to the vertebral artery, reversal of flow in the vertebral artery can occur, resulting in the theft of blood from the posterior circulation. When the upper limb is exercised, blood is diverted away from the brain to the arm. Cerebral symptoms (dizziness, syncope, etc...) depend on the integrity of collateral intracranial flow (PCOMs).

Subclavian Steal is almost always caused by atherosclerosis (98%), but other very testable causes include Takayasu Arteritis, Radiation, Preductal Aortic Coarctation, and Blalock-Taussig Shunt. In an adult they will show atherosclerosis. If they show a teenager / 20 year old it's gonna be Takayasu. Case books love to show this as an angiogram, and I think that's the most likely way the CORE will show it. They could also show a CTA or MRA although I'd say that is less likely.

Aortic Aneurysm and Vasculitis: *Will be discussed in the Vascular Chapter.*

Congenital Heart Disease

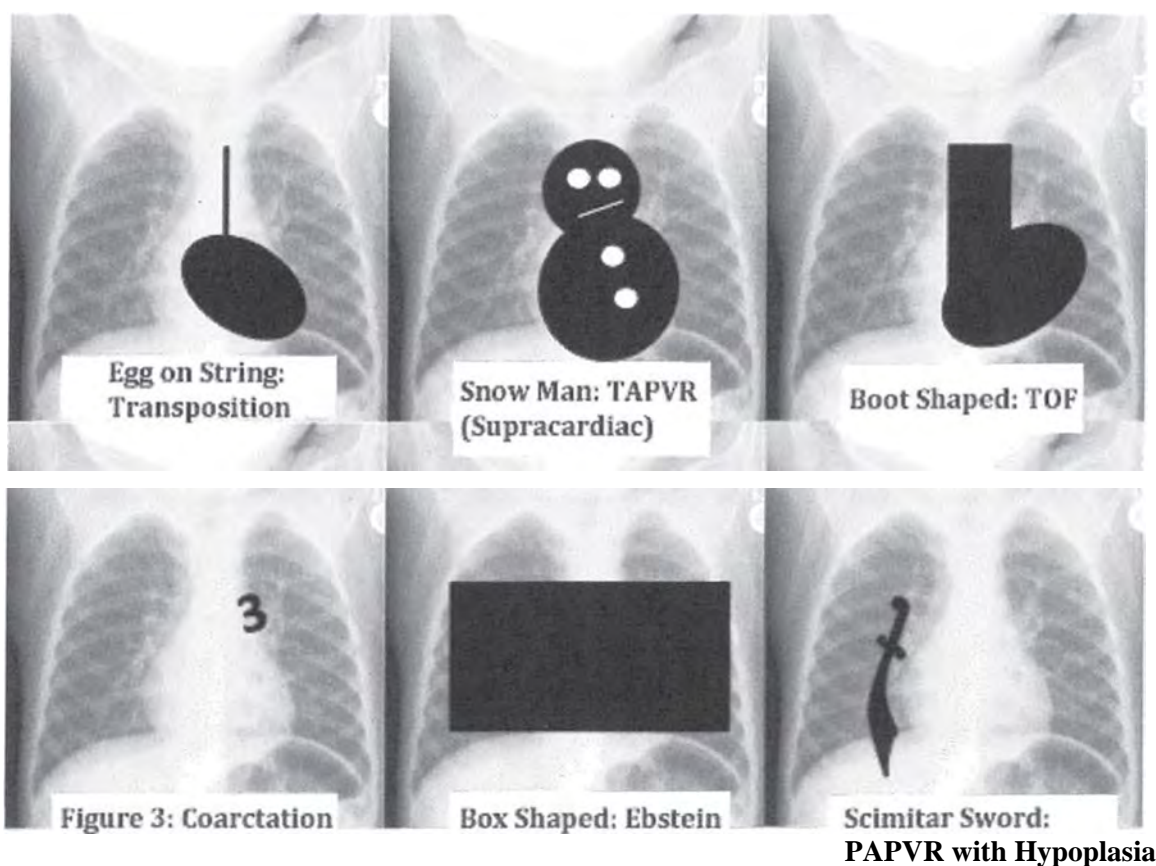
An extremely high yield and confusing topic which dinosaur Radiologists love to ask questions about on CXR. Obviously, this is stupid since you could only add confusion to a bad situation by suggesting a diagnosis on CXR instead of waiting for ECHO or MRI. Having said that, the next section will attempt to provide a methodology for single answers on CXR cases.

My thoughts on multiple choice questions regarding congenital heart is that they will come in 3 flavors: (A) Aunt Minnie, (B) Differentials with crappy distractors, and (C) Associations / Trivia.

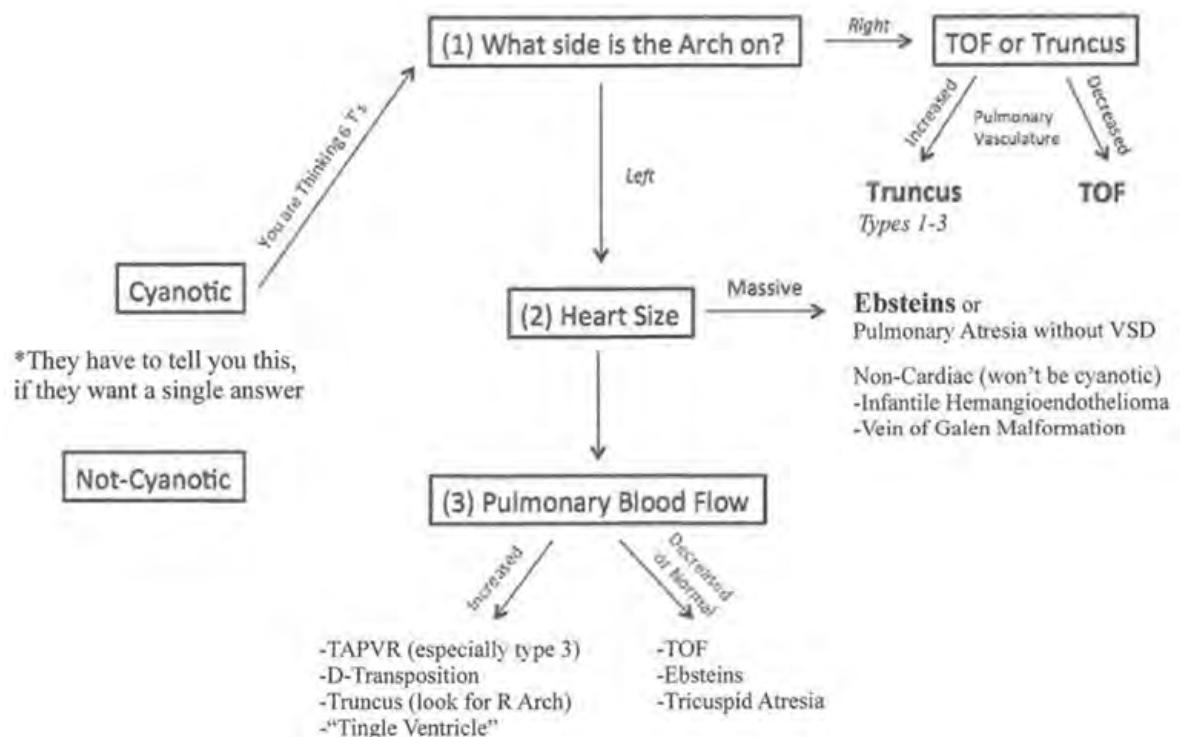
Aunt Minnies / Differentials:

There are a few congenital heart cases that are Aunt Minnies, or easily solvable (most are differential cases). Bottom line is that if they want a single answer they will have to show you either an Aunt Minnie or a differential case, with crappy distractors.

With regard to straight-up Aunt Minnies, I think the usual characters that most third year medical students memorize are fair game.



The easily solvable ones will be shown as a right arch with the associations of **Truncus** (*more closely associated*) and **TOF** (*more common overall*). Or, they will show you the big box heart and want Ebsteins (which is an Aunt Minnie). Another classic trick with regard to the big box heart is non-cardiac causes of high output failure (Infantile Hemangioendothelioma and Vein of Galen Malformation). The remaining cyanotic syndromes basically look the same, so the questions must be either (a) crappy distractors (none of the others are cyanotic, etc...), or (b) trivia (which is more likely).



With regard to identifying bad distractors I think the easiest way is the cyanotic vs not cyanotic disorders. They literally must tell you the kid is cyanotic, otherwise there is no way to know.

Cyanotic	Not Cyanotic
TOF	ASD
TAPVR	VSD
Transposition	PDA
Truncus	PAPVR
Tricuspid Atresia	Aortic Coarctation (adult type - post ductal)

There are a few other key differentials that may make it easier to weed out bad distractors, or get “which of the following do NOT” questions.

CHF in Newborn	Survival dependent on admixture: PDA, VSD, PDA - Cyanotics	Small Heart DDx
TAPVR (Infracardiac type "III")	TAPVR (has PFO)	Adrenal Insufficiency (Addisons)
Congenital Aortic or Mitral Stenosis	Transposition	Cachectic State
Left Sided Hypoplastic Heart	TOF (has VSD)	Constrictive Pericarditis
Cor Triatriatum	Tricuspid Atresia (has VSD)	
Infantile (pre-ductal) Coarctation	Hypoplastic Left	

Trivia and Associations:

VSD: The **most common congenital heart disease**. There are several types with Membranous (*just below the aortic valve*) being the most common (70%). **Outlet subtypes (infundibulum) must be repaired** as the right coronary cusp prolapses into the defect. On CXR we are very nonspecific (big heart, increased vasculature, small aortic knob). They could ask or try and show **splaying of the carina** (from big left atrium). About 70% of the small ones close spontaneously.

PDA: The PDA normally closes around 24 hours after birth (functionally), and anatomically around 1 month. A PDA should make you say three things (1) **Prematurity**, (2) **Maternal Rubella**, (3) **Cyanotic Heart Disease**. CXR is nonspecific (big heart, increased pulmonary vasculature, large aortic arch "ductus bump"). You can close it or keep it open with meds.

ASD: Several types with the Secundum being the most common (50-70%). The larger subtype is the Primum, (results from an endocardial cushion defect), is more likely to be symptomatic. Only Secundums may close without treatment (Primum, AV Canal, Sinus Venosus will not). Primums are not amendable to device closure because of proximity to AV valve tissue. On CXR, if it's small it will show nothing, if it's large it will be super nonspecific (big heart, increased vasculature, and small aortic knob). It's more common in female.

- **When I say hand/thumb defects + ASD, you say Holt Oram**
- * **When I say ostium primum ASD (or endocardial cushion defect), you say Downs**

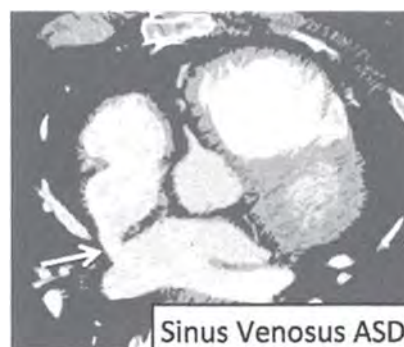
AV Canal: Also referred to as an endocardial cushion defect. They happen secondary to a deficient development of a portion of the atrial septum, a portion of the inter-ventricular septum, and the AV valves. **Strong association with Downs.** You can't use closure devices on these dudes either. Surgical approach and management is complex and beyond the scope of this text.

PAPVR: Partial anomalous pulmonary venous return, is defined as one (or more) of the four pulmonary veins draining into the right atrium. It is often of mild or no physiologic consequence. It is often **associated with ASDs (secundum and sinus venosus types).**

- * **When I say Right Sided PAPVR, you say Sinus Venosus ASD**

- o *RUL: SVC association with sinus venosus type ASD*

- * **When I say Right Sided PAPVR + Pulmonary > Hypoplasia, you say Scimitar Syndrome**



TAPVR: A cyanotic heart disease characterized by all of the pulmonary venous system draining to the right side of the heart. A large PFO or less commonly ASD is required for survival (this is a high yield and testable point). There are 3 types, but only two are likely to be tested (cardiac type II just doesn't have good testable features). All 3 types will cause increased pulmonary vasculature, but type 3 is famous for a full on pulmonary edema look in the newborn.

- * Type 1: Supracardiac:
 - o Most Common Type
 - o Veins drain above the heart, **gives a snowman appearance.**
- * Type 2: Cardiac
 - o Second Most Common Type
- * Type 3: Infracardiac
 - o Veins drain below the diaphragm (hepatic veins or IVC)
 - o **Obstruction on the way back through diaphragm is common and causes a full on pulmonary edema look**

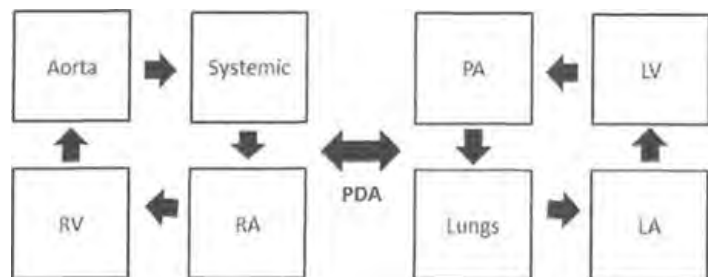
Key Points on TAPVR:

- * Supracardiac Type = Snowman
- * Infracardiac Type = Pulmonary Edema in Newborn
- * Large PFO (or ASD) needed to survive
- * Asplenia - 50% of asplenia patients have congenital heart issues, of those nearly 100% include TAPVR, (85% have additional endocardial cushion defects).

Transposition: This is the most common cause of cyanosis during the first 24 hours. It is seen most commonly in infants of diabetic mothers. The basic idea is that aorta arises from the right ventricle and the pulmonary trunk from the left ventricle (*ventriculararterial discordance*).

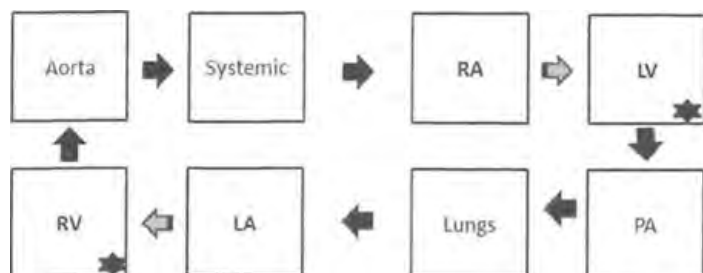
Just like TAPVR survival depends of an ASD, VSD, or PDA (*most commonly VSD*). There are two flavors: D & L. The D type only has a PDA connecting the two systems. Where as the L type is “Lucky” enough to be compatible with Life.

D-Transposition: Classic radiographic appearance is the “egg on a string”. Occurs from discordance between the ventricles and the vessels. The intra-atrial baffle (Mustard or Senning procedure) is performed to fix them



In D-Transposition the ductus may be the only connection between the two systems, which would otherwise be separate (and not compatible with life)

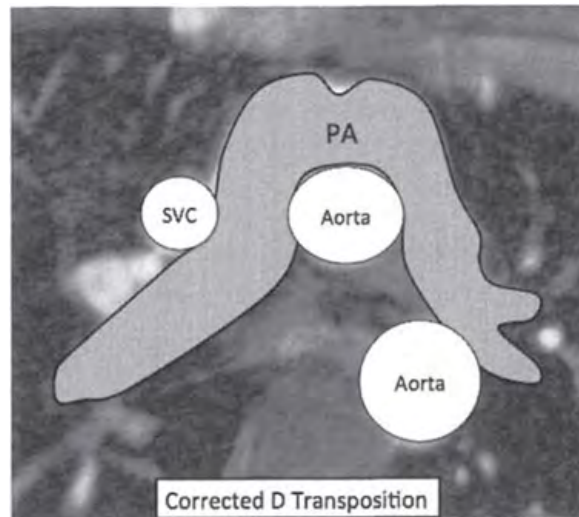
L-Transposition: The L type is “Lucky” enough to be congenitally corrected. This occurs from a “double discordance” where the atrium hooks up with the wrong ventricle and the ventricle hooks up with the wrong vessels.



In L-Transposition Transposition of the great vessels occurs with inversion of the ventricle, leading to “congenital correction.” No PDA is needed.

A **corrected D-transposition** has a very characteristic appearance, lending itself to an Aunt Minnie-type question.

The PA is draped overtop the Aorta, which occurs after a surgeon has performed to “*LeCompte Maneuver*” — sounds French so must be high yield.



Tetralogy of Fallot (TOF): The *most common cyanotic heart disease*. Describes 4 major findings; (1) VSD, (2) RVOT Obstruction - often from valvular obstruction, (3) Overriding Aorta, (4) RV hypertrophy (develops after birth). The degree of severity in symptoms is related to how bad the RVOT obstruction is. If it's mild you might even have a “pink tet” that presents in early adulthood. This is called a pentaology of Fallot if there is an ASD. Very likely to have a right arch.

Surgically it's usually fixed with primary repair. The various shunt procedures (Blalock-Taussig being the most famous) is only done if the kid is inoperable or to bridge until primary repair.

Truncus Arteriosus: Cyanotic anomaly where there is a single trunk supplying both the pulmonary and systemic circulation, not of a separate aorta and a pulmonary trunk. It almost always has a VSD, and is closely associated with right arch. **Associated with CATCH-22** genetics (DiGeorge Syndrome).

Coarctation: Narrowing of the aortic lumen. This comes in two flavors:

- (1) Infantile (Pre-ductal) - these guys can have pulmonary edema
- (2) Adult (Ductal)

OK, things to know: Strong Association with **Turners Syndrome** (15-20%). **Bicuspid Aortic valve** is the most common associated defect (80%). They have more **berry aneurysms**. **Figure 3 sign** (appearance of CXR). *

* **Rib Notching:** most often involves **4th - 8th ribs**. It does NOT involve the 1st and 2nd because those are fed by the costocervical trunk.

Hypoplastic Left Heart: Left ventricle and aorta are hypoplastic. They present with pulmonary edema. **Must have an ASD** or large PFO. They also typically have a large PDA to put blood in their arch. Strongly associated with aortic coarctation and endocardial fibroelastosis.

Cor Triatriatum Sinistrum: This is a very rare situation where you have an **abnormal pulmonary vein draining into the left atrium** (*sinistrum meaning left*) with an unnecessary fibromuscular membrane that causes a sub division of the left atrium. This **creates the appearance of a tri-atrium heart**. This can be a cause of unexplained pulmonary hypertension in the peds setting. Basically it acts like mitral stenosis, and can cause **pulmonary edema**. The outcomes are often bad (fatal within two years), depending on surgical intervention and associated badness.



Cor Triatriatum Sinistrum

Ischemic Heart Disease

Imaging regarding ischemic heart disease is going to fall into two modalities; cardiac MR, and Nuclear. Cardiac MRI currently offers the most complete evaluation of ischemic heart disease.

Myocardial infarction typically is initiated by rupture of an unstable coronary atherosclerotic plaque, leading to abrupt arterial occlusion. The **wave front of necrosis always starts subendocardial and progresses to the subepicardium**. The ischemic necrosis will affect not just the myocardium but also blood vessels. The destruction of small capillaries will not allow contrast to the area of injury. This is termed “microvascular obstruction” and manifests as islands of dark signal in an ocean of delayed enhancement. The presence of microvascular obstruction is an independent predictor of death and adverse LV remodeling.

Testable Vocab:

- * *Stunned Myocardium*: After an Acute Injury (ischemia or reperfusion injury), dysfunction of myocardium persists even after restoration of blood flow (can last days to weeks). A perfusion study will be normal, but the contractility is crap.
- * *Hibernating Myocardium*: This is a more chronic process, and the result of severe CAD causing chronic hypoperfusion. You will have areas of **decreased perfusion and decreased contractility** even when resting. Don’t get it twisted, **this is not an infarct. On a FDG PET, this tissue will take up tracer more intensely than normal myocardium, and will also demonstrate redistribution of thallium**. This is reversible with revascularization.
- * *Scar*: This is dead myocardium. It will not squeeze normally, so you’ll have abnormal wall motion. It’s not a zombie. It will not come back to life with revascularization.

Stunned	Hibernating	Infarct / Scar
Wall Motion Abnormal	Wall Motion Abnormal	Wall Motion Abnormal
Normal Perfusion (Thallium or Sestamibi)	Abnormal Fixed Perfusion	Abnormal Fixed Perfusion
	Will Redistribute with Delayed Thallium and will take up FDG	Will NOT Redistribute with Delayed Thallium, will NOT take up FDG
Associated with acute MI	Associated with chronic high grade CAD	Associated the chronic prior MI

Delayed imaging: It works for two reasons: (1) Increased volume of contrast material distribution in acute myocardial infarction (and inflammatory conditions) (2) Scarred myocardium washes out more slowly. It is **done using an inversion recovery' technique** to null normal myocardium, followed by a gradient echo. T1 shortening from the Gd looks bright ("Bright is Dead").

Why stress imaging is done: Because coronary arteries can auto-regulate, a stenosis of **85%** can be asymptomatic in a resting state. So demand is increased (by exercise or drugs) making a **45%** stenosis significant. An inotropic stress agent (dobutamine) is used for wall motion, and a vasodilator (adenosine) is used for perfusion analysis.

MRI in Acute MI: Cardiac MRI can be done in the first 24 hours post MI (if the patient is stable). Late gadolinium enhancement will reflect size and distribution of necrosis. Characteristic pattern is a **zone of enhancement that extends from the subendocardium toward the epicardium in a vascular distribution**. Microvascular obstruction will present as islands of dark signal in the enhanced tissue (as described above), and this represents an acute and subacute finding. **Microvascular obstruction is NOT seen in chronic disease** as these areas will all turn to scar eventually. In the acute setting (1 week) injured myocardium will have increased T2 signal, which can be used to estimate the area at risk (*T2 Bright - Enhanced = Salvageable Tissue*).

Acute vs Chronic MI:

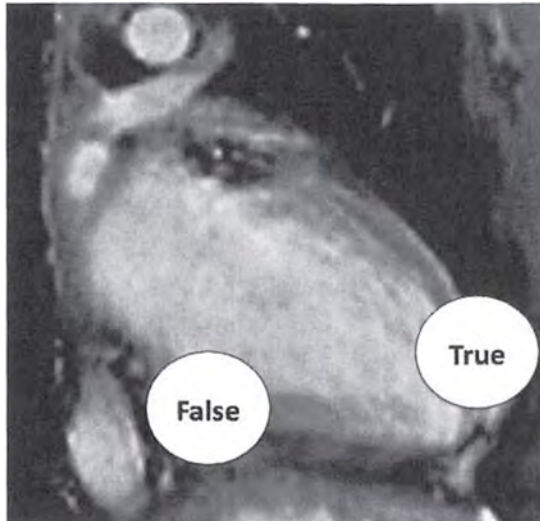
- * *Both have delayed enhancement*
- * *If the infarct was transmural and chronic you may have thinned myocardium*
- * *Acute will have normal thickness (chronic can too but shouldn't for the purposes of MC tests.*
- * *T2 signal from edema may be increased in the acute setting*
- * *You won't see Microvascular Obstruction in Chronic*

Microvascular Obstruction: Islands of dark tissue in an ocean of late Gd enhancement. These indicate microvascular obliteration in the setting of an acute infarct. The Gd is unable to get to these regions even after the restoration of epicardial blood flow. Microvascular obstruction is a **poor prognostic finding**, associated with lack of functional recovery. **It's NOT seen in chronic infarct.**



Ventricular Aneurysm: This is rare (5%), but can occur as the result of MI. The question is always true vs false:

- * *True:* Mouth is wider than body. Myocardium is intact. Usually anterior-lateral wall.
- * *False:* Mouth is narrow compared to body. Myocardium is NOT intact (pericardial adhesions contain the rupture). Usually posterior-lateral wall. Higher risk of rupture.



False Aneurysms Are Usually Posterior-Lateral
True Aneurysms Are Usually Anterior-Lateral

Viability - You can grade this based on % of transmural thickness involved in the infarct.

- <25%: likely to improve with PCI
- 25-50%: may improve
- 50-100%: unlikely to recover function

What is the timing on the bad sequelae of an MI?

Dressier Syndrome	4-6 weeks
Papillary Muscle Rupture	2-7 Days
Ventricular Pseudoaneurysm	3-7 Days
Ventricular Aneurysm	Months - Requires remodeling and thinning.
Myocardial Rupture	Within 3 Days (50% of the time)

Non-Ischemic Disease

Dilated Cardiomyopathy: Defined as dilatation with an end diastolic diameter greater than 55mm, with a decreased EF. Can be idiopathic, ischemic, or from a whole list of other random crap (Alcohol, Doxorubicin, Chagas, etc...). The ischemic variety may show subendocardial enhancement. The **idiopathic variety will show** either no enhancement or **linear midmyocardial enhancement**. There is often an association with mitral regurgitation due to dilation of the mitral ring.

Restrictive Cardiomyopathy: Basically anything that causes a decrease in diastolic function. Can be the result of myocardium replaced by fibrotic tissue (endocardial fibroelastosis), infiltration of the myocardium (Amyloidosis), or damage by iron (hemochromatosis). **The most common cause is actually amyloid.**

- * Amyloidosis: Deposits in the myocardium causes abnormal diastolic function with biatrial enlargement, concentric thickening of the left ventricle and reduced systolic function of usually both ventricles. Seen in 50% of cases of systemic amyloid. Has a terrible prognosis. You can sometimes see late Gd enhancement over the entire subendocardial circumference.

Amyloid Classic Scenario: A **long TI is needed** (like 350 milliseconds, normal would be like 200). TI will be so long that the blood pool may be darker than the myocardium. **Buzzword “difficult to suppress myocardium”.**

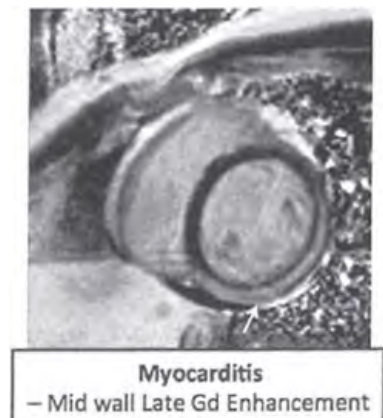
- * Eosinophilic Cardiomyopathy : **Bilateral Ventricular thrombus** is the classic phrase / buzzword

Constrictive Pericarditis: Historically this used to be TB or Viral. Now the most common cause is iatrogenic secondary to CABG or radiation. On CT the pericardium is too thick (>0.4cm), and if it's calcified that is diagnostic. Calcification is usually largest over the AV groove. “Sigmoidization” is seen on SSFP cine imaging: The ventricular septum moves toward the left ventricle in a wavy pattern during early diastole (“**Diastolic Bounce**”).

This vs That: Constrictive vs Restrictive Cardiomyopathy:

- * *Pericardium is usually thickened in constrictive*
- * *Diastolic septal bounce is seen in constrictive (Sigmoidization of the septum).*

Myocarditis: Inflammation of the heart can come from lots of causes (*often viral i.e. Coxsackie virus*). The late Gd enhancement follows a non-vascular distribution preferring the **lateral free wall**. The **pattern will be epicardial or mid wall (NOT subendocardial)**.

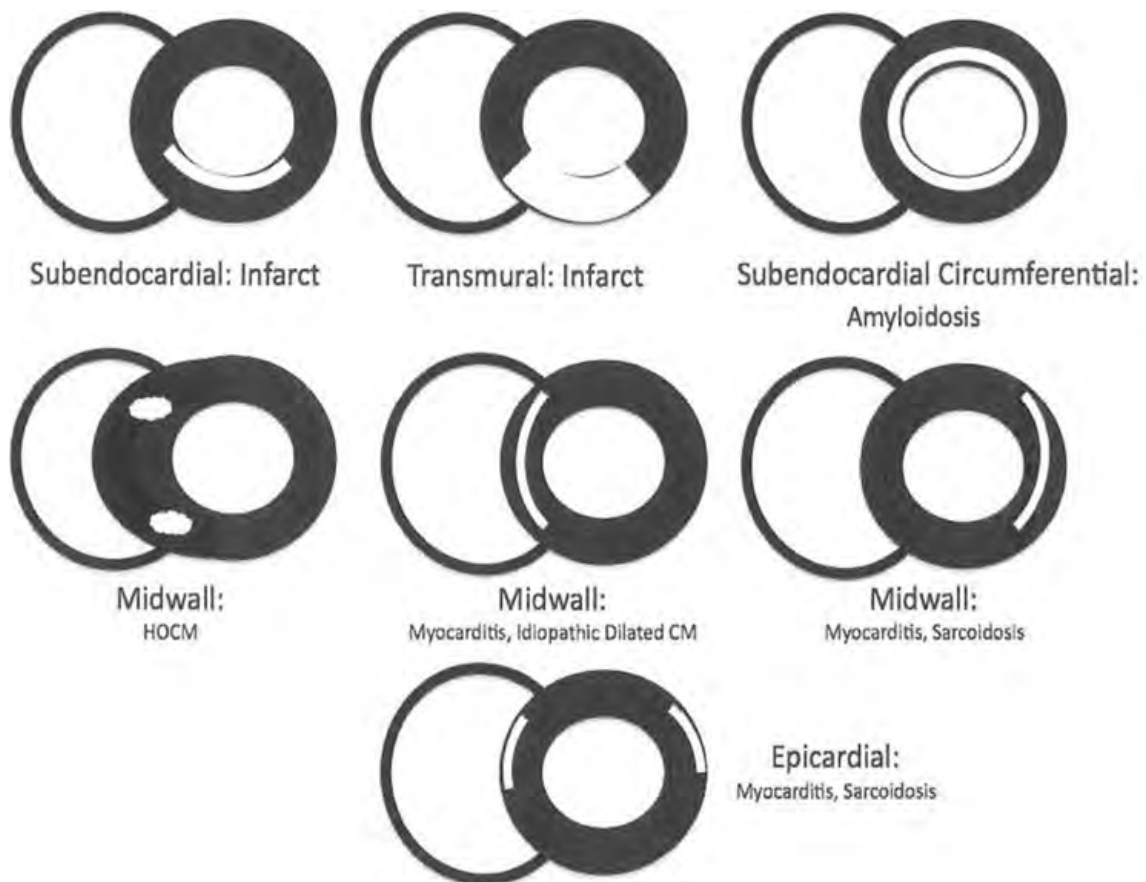


Sarcoidosis: Cardiac involvement is seen in 5% of Sarcoidosis cases, and is associated with an increased risk of death. Signal in both T2 and early Gd (as well as late Gd) will be increased. Late Gd pattern may be middle and epicardial in a non-coronary distribution.

Focal wall thickening from edema can mimic hypertrophic cardiomyopathy. It often involves the septum. The RV and papillaries are RARELY affected.

Takotsubo Cardiomyopathy - A takotsubo is a Japanese Octopus trap, which looks like a pot with a narrow mouth and large round base. The octopus will go into the pot, but then can't turn around and get out. A condition with Chest pain and EKG changes seen in post menopausal women after they either break up with their boyfriend, win the lottery, or some other stressful event has been described with the shape of the ventricle looking like a takotsubo. There is **transient akinesia or dyskinesia of the left ventricular apex without coronary stenosis. Ballooning of the left ventricular apex is a buzzw ord.** No delayed enhancement.

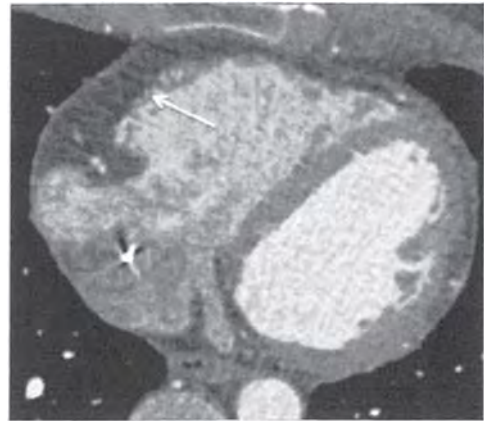
Classic Patterns of Delayed Enhancement:



Genetic Conditions

Arrhythmogenic Right Ventricular

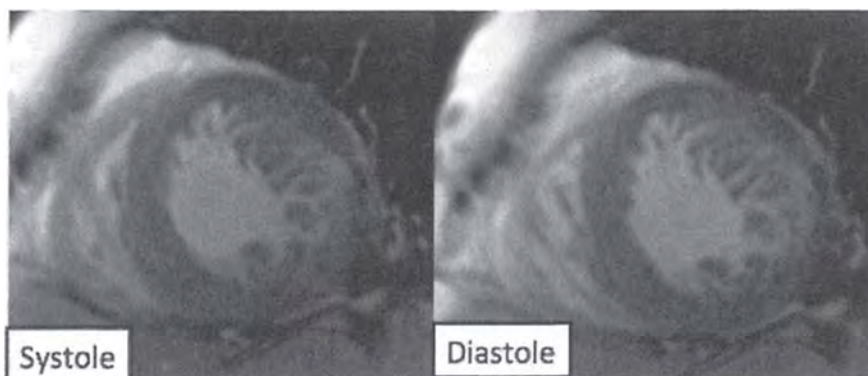
Cardiomyopathy (ARVC): Characterized by fibrofatty degeneration of the RV leading to arrhythmia and sudden death. Features include dilated RV with reduced function, **fibrofatty replacement of the myocardium**, and normal LV. People use this major/minor criteria system that includes a bunch of EKG changes that no radiologist could possibly understand (if they are stupid enough to ask just say left bundle branch block). Watch out for the use of fat sat to demonstrate the fat in the RV wall.



ARVC -Dilated RV, with Fat in the wall

Hypertrophic Cardiomyopathy: Abnormal hypertrophy (from disarray of myofibrils) of the myocardium that compromises diastole. There are multiple types but the one they are going to show is asymmetric hypertrophy of the intraventricular septum. The condition is a cause of sudden death. There is a subgroup which is associated with LVOT obstruction (“hypertrophic obstructive cardiomyopathy”). Venturi forces may pull the anterior leaflet of the mitral valve into the LVOT (**SAM** - Systolic Anterior Motion of the Mitral Valve). Patchy midwall **delayed enhancement of the hypertrophied muscle** may be seen, as **is an independent risk factor for sudden death**.

Noncompaction: Left ventricular noncompaction is a uncommon congenital cardiomyopathy that is the result of loosely packed myocardium. The left ventricle has a spongy appearance with increased trabeculations and deep intertrabecular recesses. As you might expect these guys get heart failure at a young age. Diagnosis is based of a ratio of non compacted end-diastolic myocardium to compacted end-diastolic myocardium of more than 2.3:1.



Noncompaction – Spongy LV, with no myocardial thickening

Muscular Dystrophy - Becker (mild one) and Duchenne (severe one) are X-linked neuromuscular conditions. They have biventricular replacement of myocardium with connective tissue and fat (delayed Gd enhancement in the midwall). They often have dilated cardiomyopathy. Just think **kid with dilated heart and midwall enhancement**.

Tumors

Mets: Thirty times more common than a primary malignancy. The **pericardium is the most common site** affected (by far). The most common manifestation is a pericardial effusion (second most common is a pericardial lymph node). Melanoma may involve the myocardium.

Angiosarcoma: Most common primary malignant tumor of the heart in adults. They like the RA and tend to involve the pericardium. They often cause right sided failure and/or tamponade. They are bulky and heterogenous. Buzzword is “sun-ray” appearance which describes enhancement appearance of the diffuse subtype as it grows along the perivascular spaces associated with the epicardial vessels.

Left Atrial Myxoma: Most common primary cardiac tumor in adults (rare in children). They are associated with MEN syndromes, and Blue Nevi (Carney Complex). They are most often **attached to the interatrial septum**. They may be calcified. They may prolapse through the mitral valve. They **will enhance with Gd** (important discriminator from a thrombus).

Tumor vs Thrombus:

Cardiac MRI is the way to tell.

- Tumor will enhance
- Thrombus will NOT enhance.

Rhabdomyoma: Most common fetal cardiac tumor. It is a hamartoma. They prefer the **left ventricle**. Associated with **tuberous sclerosis**. Most tumors will regress spontaneously (those NOT associated with TS are actually less likely to regress).

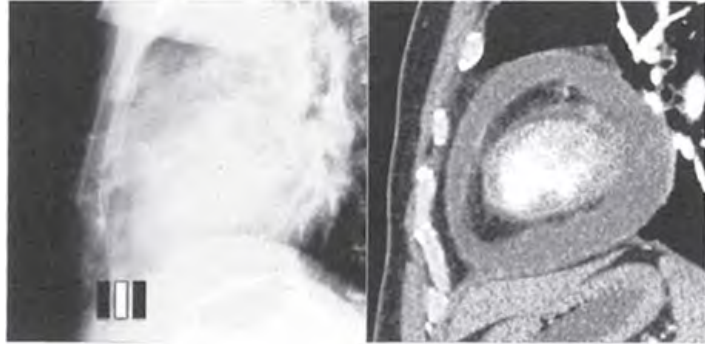
Fibroma: Second most common cardiac tumor in childhood. They like the IV septum, and are dark / dark on T1/T2. They enhance very brightly on perfusion and late Gd.

Fibroelastoma: Most common neoplasm to involve the **cardiac valves** (80% aortic or mitral). They are highly mobile on SSFP Cine. Systemic emboli are common (especially if they are on the left side).

Pericardial Disease

The pericardium is composed of two layers (visceral and parietal), with about 50cc of fluid normally between the layers.

Pericardial Effusion: Basically more than 50cc between the pericardial layers. This can be from lots and lots of causes - renal failure (uremia) is probably the most common. For the purpose of multiple choice tests you should think about Lupus, and Dressier Syndrome (inflammatory effusion post MI). On CXR they could show this 3 ways: (1) Normal Heart on Comparison, Now Really Big Heart (2) Giant Water Bottle Heart, (3) Lateral CXR with two lucent lines (epicardial and pericardial fat) and a central opaque line (pericardial fluid) - the so called “**oreo cookie sign**.”



Pericardial Effusion : "Oreo Cookie Sign"

Cardiac Tamponade: Pericardial effusion can cause elevated pressure in the pericardium and result in compromised filling of the cardiac chambers (atria first, then ventricles). This can occur with as little as 100cc of fluid, as the **rate of accumulation is the key factor** (chronic slow filling gives the pericardium a chance to stretch). The question is likely related to **short-axis imaging during deep inspiration showing flattening or inversion of the intraventricular septum toward the LV**, a consequence of augmented RV filling. Another indirect sign that can be shown on CT is reflux of contrast into the IVC and azygos system.

Pericardial Cysts: Totally benign incidental finding. Usually seen on the right cardiophrenic sulcus. They do not communicate with the pericardium. Rarely they can get infected or hemorrhage. **They will show you an ROI measuring water density along the right cardiophrenic sulcus, and this will be the answer.**

Congenital / Acquired Absence: Absence of the pericardium may involve all or part of the pericardium. **Most commonly** the absence is partial and **involves the pericardium over the left atrium and adjacent pulmonary artery**. The *left atrial appendage is the most at risk to become strangulated*. When the left pericardium is absent the heart shifts towards the right. They could show you a CT or MRI with the heart contacting the left chest wall, and want you to infer partial absence. Another piece of trivia is that cardiac herniation and volvulus can occur in patients who undergo extrapleural pneumonectomy (herniation only occurs if the lung has also been removed).

Constrictive Pericarditis - *Discussed above under non-ischemic.*

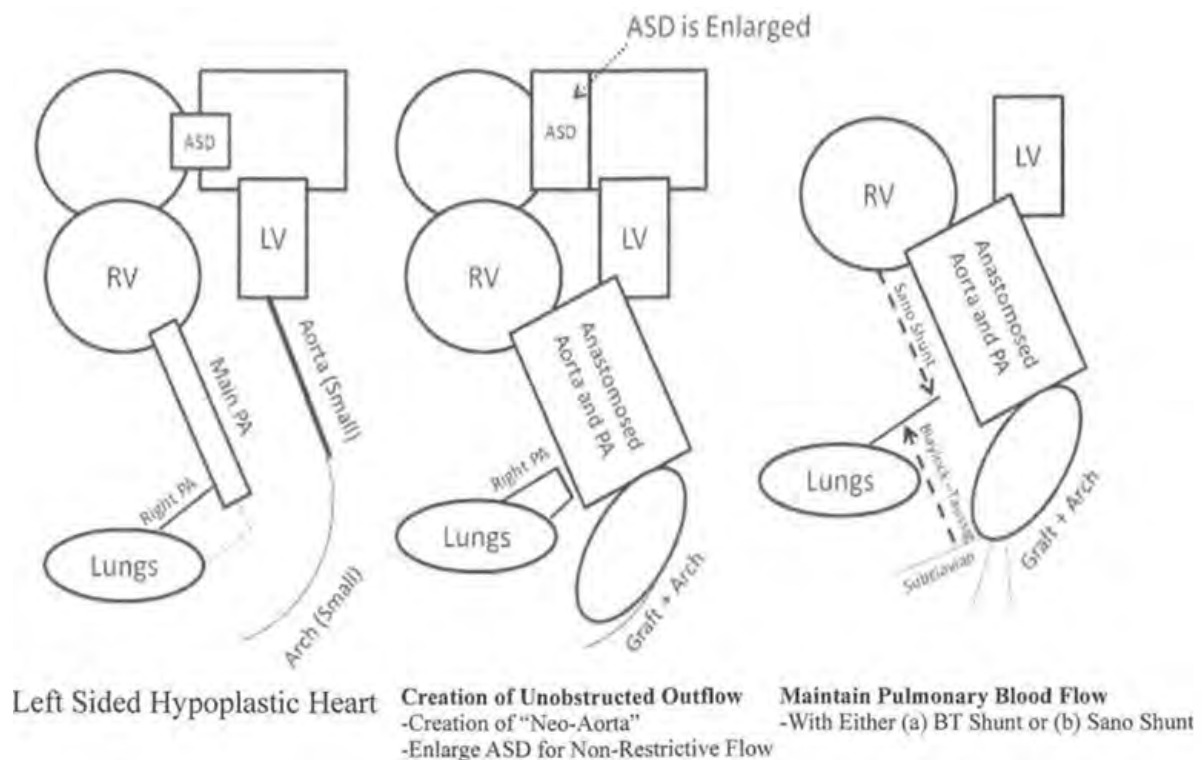
Cardiac Surgeries:

Palliative Surgery for the Hypoplastic Left Heart: Surgery for Hypoplasts is not curative, and is instead designed to extend the life (prolong the suffering) of the child. It is done in a 3 stage process, to protect the lungs and avoid right heart overload:

- (1) Norwood or Sano - within days of birth
- (2) Glenn - at 3- 6 months
- (3) Fontan at 1 Zi to 5 years

Norwood: The goal of the surgery is to create an unobstructed outflow tract from the system icventricle. So the tiny native aorta is anastomosed to the pulmonary trunk, and the arch is augmented with a graft (or by other methods). The ASD is enlarged to create non restrictive atrial flow. A Blaylock-Taussig Shunt (see below) is used between the right Subclavian and right PA. The ductus is removed as well to prevent over shunting to the lungs. Apparently, when this goes bad it's usually from issues related to damage of the coronary arteries or **over shunting of blood to the lungs (causing pulmonary edema)**. As a point of trivia, sometimes the thymus is partially removed to get access.

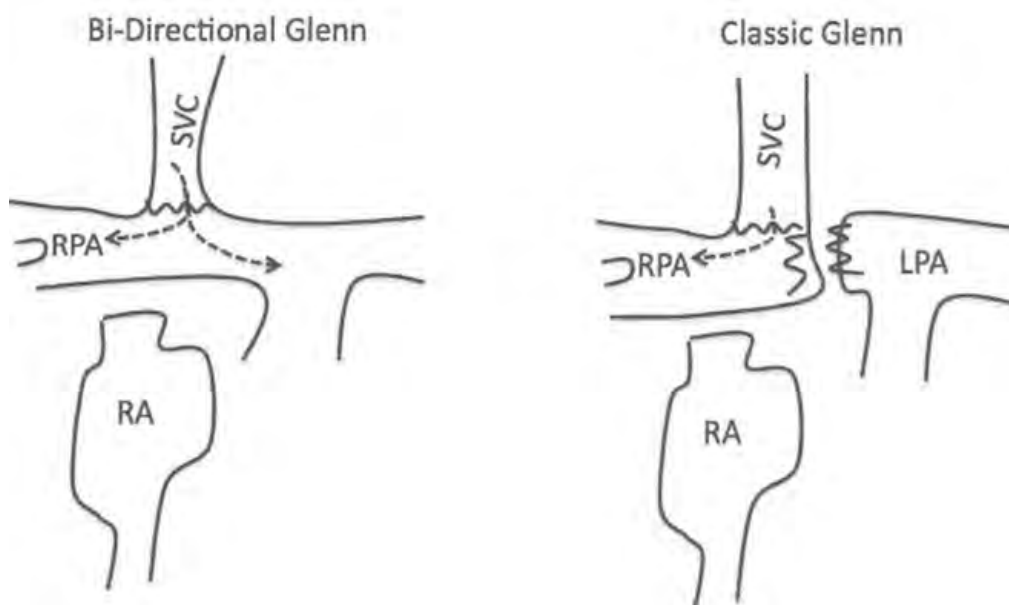
Norwood Procedure



Sano: Same as the Norwood, but instead of using a Blalock-Taussig shunt a conduit is made connecting the right ventricle to the pulmonary artery. The disadvantage of the BT Shunt undergoes a steal phenomenon (diverted to low pressure pulmonary system).

Classic Glenn: Shunt between the SVC and right pulmonary artery (end-to-end), with the additional step of sewing the proximal end of the Right PA closed with the goal of reducing right ventricular work, by diverting all venous return straight to the lung (right lung).

Bi-Directional Glenn: Shunt between the SVC and the right pulmonary artery (**end-to-side**). The RPA is left open, letting blood flow to both lungs. This procedure can be used to address right sided heart problems in general, and is also step two in the palliative hypoplastic series. If it's being used as step two the previously placed Blalock Taussig Shunt or Sano shunt will come down as the Glenn will be doing its job of putting blood in the lungs.



Fontan Operation: Used for Hypoplastic Hearts. The old school Fontan consisted of a classic Glenn (SVC to RPA), closure of the ASD, and then placing a shunt between the Right atrium to the Left PA. The idea is to let blood return from systemic circulation to the lungs by passive flow (no pump), and turn the right ventricle (the only one the kid has) into a functional left ventricle. There are numerous complications including right atriomegaly with resulting arrhythmias, and plastic bronchitis (they cough up “casts of the bronchus” that look like plastic).

Other Surgeries:

Classic Blalock Taussig Shunt: Originally developed for use with TOF. **Shunt is created between the Subclavian artery and the pulmonary artery.** It is constructed on the **opposite side** of the arch. It's apparently technically difficult and often distorts the anatomy of the pulmonary artery.

High Yield Point:

Glenn = Vein to Artery
(SVC to Pulmonary Artery)

Blalock Taussig = Artery to Artery
(Subclavian Artery to Pulmonary Artery)

Modified Blalock Taussig Shunt: This is a gortex shunt between the Subclavian artery and pulmonary artery, and is performed on the **SAME SIDE as the arch**. It's easier to do than the original.

Pulmonary Artery Banding: Done to reduce pulmonary artery pressure (goal is 1/3 of systemic pressure). Most common indication is CHF in infancy with anticipated delayed repair. The **single ventricle is the most common lesion requiring banding**.

Atrial Switch: Mustard and Senning procedures are used to correct transposition of the great arteries by creating a baffle within the atria in order to switch back the blood flow at the level of in-flow. The result is the right ventricle becomes the systemic ventricle, and the left ventricle pumps to the lungs. This is usually done in the first year of life.

- **Senning:** Baffle is created from the right atrial wall and atrial septal tissue WITHOUT use of extrinsic material
- * **Mustard:** Involves the resection of the atrial septum and creation of a baffle using pericardium (or synthetic material).

Rastelli Operation: Most frequency used operation for transposition, pulmonary outflow obstruction, and VSD. The procedure involves the placement of a baffle within the right ventricle diverting flow from the VSD to the aorta, essentially using the VSD as part of the LVOT. The pulmonary valve is oversewn and the conduit is inserted between the RV and the PA. The primary advantage of this procedure is the left ventricle becomes the system ventricle. The primary limitation of this procedure is that the child will be committed to multiple additional surgeries because the conduit wears out and must be replaced.

Jatene Procedure: This is another arterial switch method that involves transection of the aorta and pulmonary arteries about the valve sinuses, including the removal of the coronaries. The great arteries are switched and the coronaries are sewn into the new aorta (formerly the PA). Apparently this is very technically difficult, but the advantage is there is no conduit to go bad, and the LV is the systemic vessel.

Ross Procedure: Performed for Diseased Aortic Valves in Children. Replaces the aortic valve with the patient's pulmonary valve and replaces the pulmonary valve with a cryopreserved pulmonary valve homograft. Follow-up studies have shown interval growth of the aortic valve graft in children and infants.

Bentall Procedure: Operation involving composite graft replacement of the aortic valve, aortic root and ascending aorta, with re-implantation of the coronary arteries into the graft. This operation is used to treat combined aortic valve and ascending aorta disease, including lesions associated with Marfan syndrome

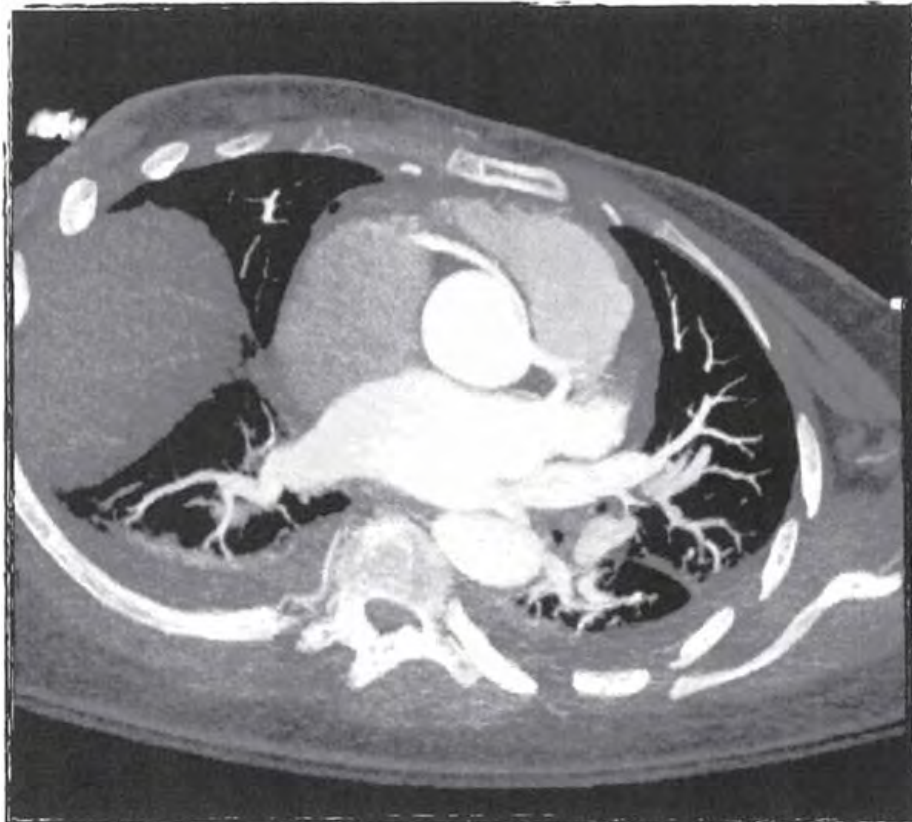
Heart Transplant Types

Orthotopic heart transplants all of the heart is removed, except the circular part of the left atrium (the part with the pulmonary veins). The donor heart is trimmed to fit into the left atrium.

Heterotopic heart transplants the recipient heart remains in place, and the donor heart is added on top. This basically creates a double heart. The advantages of this are (1) it gives the native heart a chance to recover , and (2) gives you a backup if the donor is rejected.

8 Vascular

Prometheus Lionhart, M.D.



Questions on vascular imaging tend to be image rich with a heavy focus on anatomy. Lots of collateral pathways, lots of variant anatomy, lots of testable trivia. Remember anatomy can be shown on CTA, MRA, or Angiogram.

High Yield Topics:

- Anatomy
- Acute Aortic Syndrome
- Marfans
- Vasculitis
- Carotid Doppler

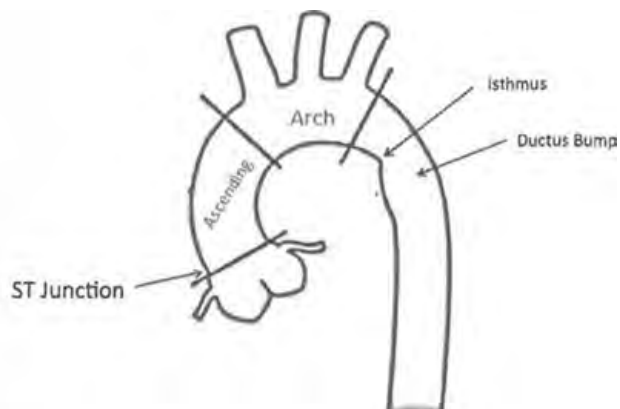
Anatomy:

Aorta: The thoracic aorta is divided anatomically into four regions; the root, the ascending aorta, the transverse aorta (arch), and the descending aorta. The “root” is defined as the portion of the aorta extending from the aortic valve annulus to the sinotubular junction. The diameter of the thoracic aorta is largest at the aortic root and gradually decreases (*average size is 3.6cm at the root, 2.4cm in the distal descending*).

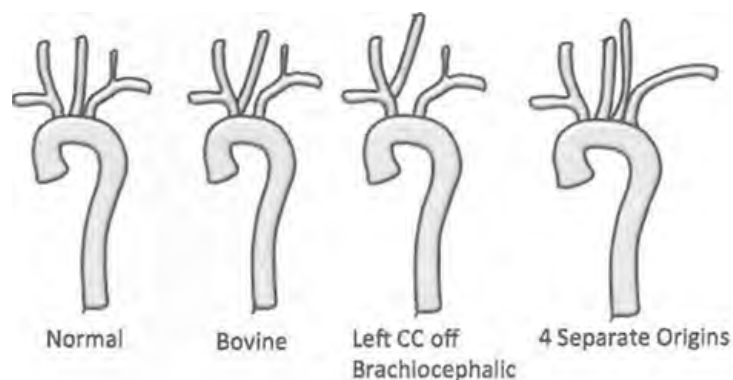
Sinuses of Valsalva: There are 3 outpouching (right, left, posterior) above the annulus that terminate at the ST Junction. The right and left coronaries come off the right and left sinuses. The posterior cusp is sometimes called the “non-coronary cusp.”

Isthmus: The segment of the aorta between the origin of the left Subclavian and the ligamentous arteriosum.

Ductus bump: Just distal to the isthmus is a contour bulge along the lesser curvature, which is a normal structure (not a pseudoaneurysm).

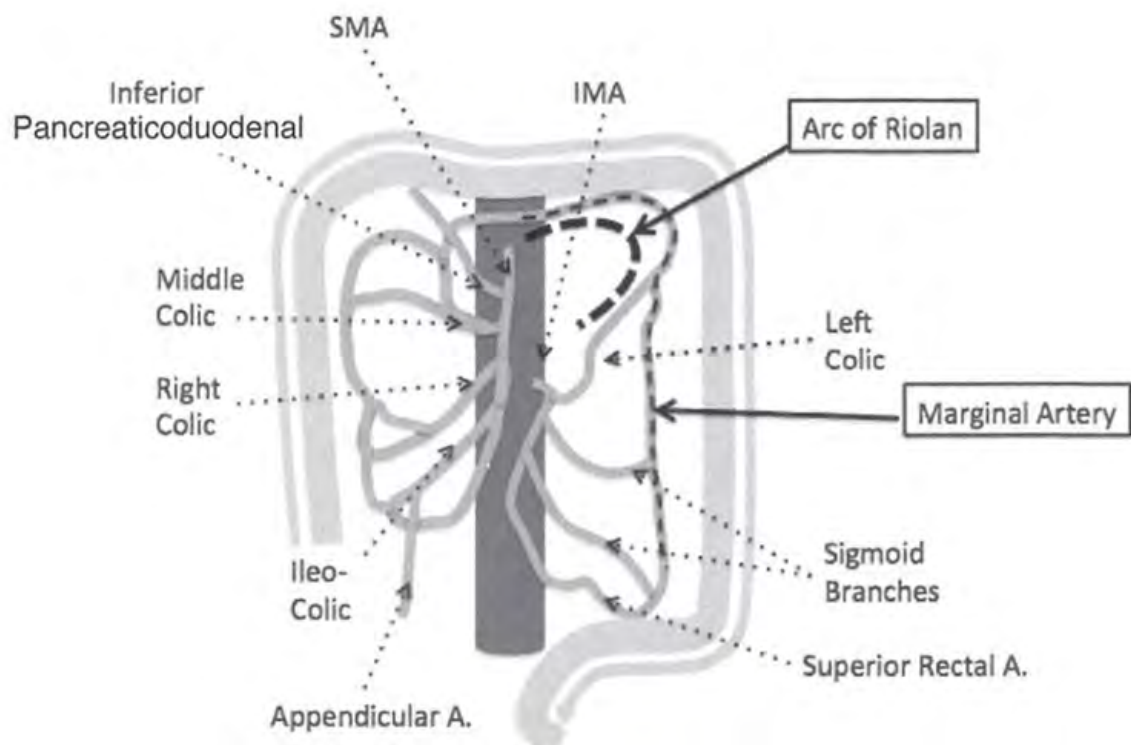


Aortic Arch Variants: There are 4 common variations: Normal (75%), Bovine Arch (15%) - common origin of brachiocephalic artery and left common carotid artery, left common carotid coming off the brachiocephalic proper (10%), and 5% of people the left vertebral artery originates separately from the arch. Branching with regards to right arch, left arch and double arch are discussed in more detail in the cardiac chapter.



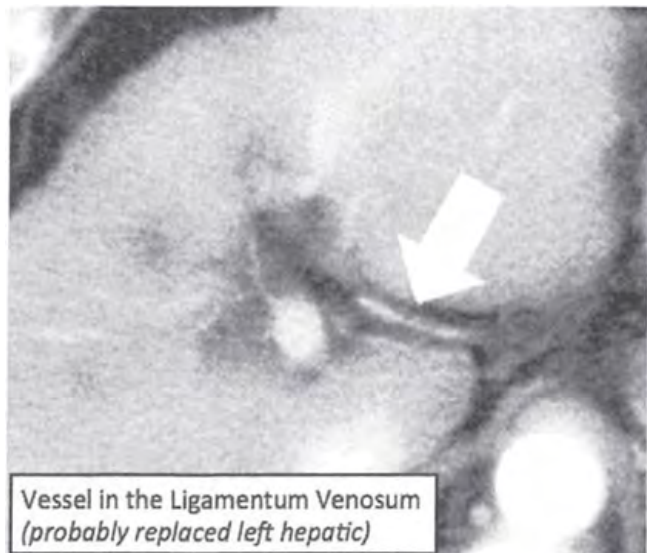
Adamkiewicz: The thoracic aorta puts off multiple important feeders including the great anterior medullary artery (Artery of Adamkiewicz) which serves as a dominate feeder of the spinal cord. This thing usually comes off on the **left side (70%) between T8-L1 (90%)**.

Mesenteric Branches - The anatomy of the SMA and IMA is high yield, and can be shown on a MIP coronal CT, or Angiogram. I think that knowing the inferior pancreaticoduodenal comes off the SMA first, and that the left colic (from IMA) to the middle colic (from SMA) make up the Arc of Rioloan are probably the highest yield facts.



Variant Hepatic Artery Anatomy - The classic branches of the common hepatic artery from the celiac artery, with the proper hepatic artery (after the GDA) and then the right and left hepatic is actually only seen in 55-60% of people. The **most common variants are the right hepatic artery replaced off the SMA** (notice I said “replaced” not accessory since there is still just one), and the left hepatic “replaced” off the left gastric.

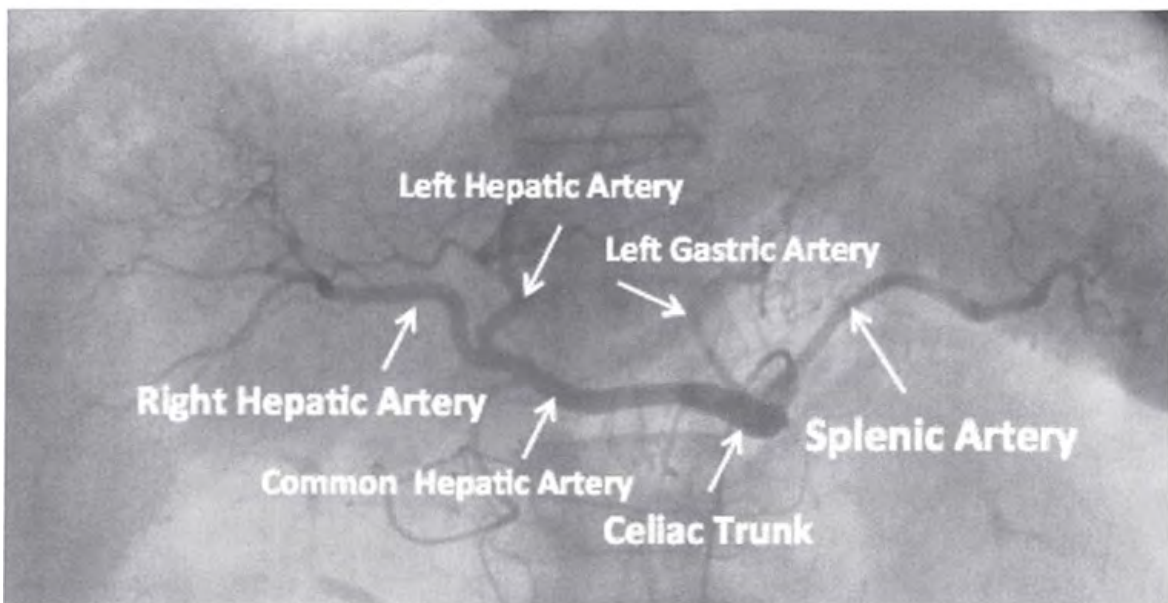
If you see a **vessel in the fissure of the ligamentum venosum** (where there is not normally a vessel), it's probably an accessory or **replaced left hepatic artery arising from the left gastric artery**.



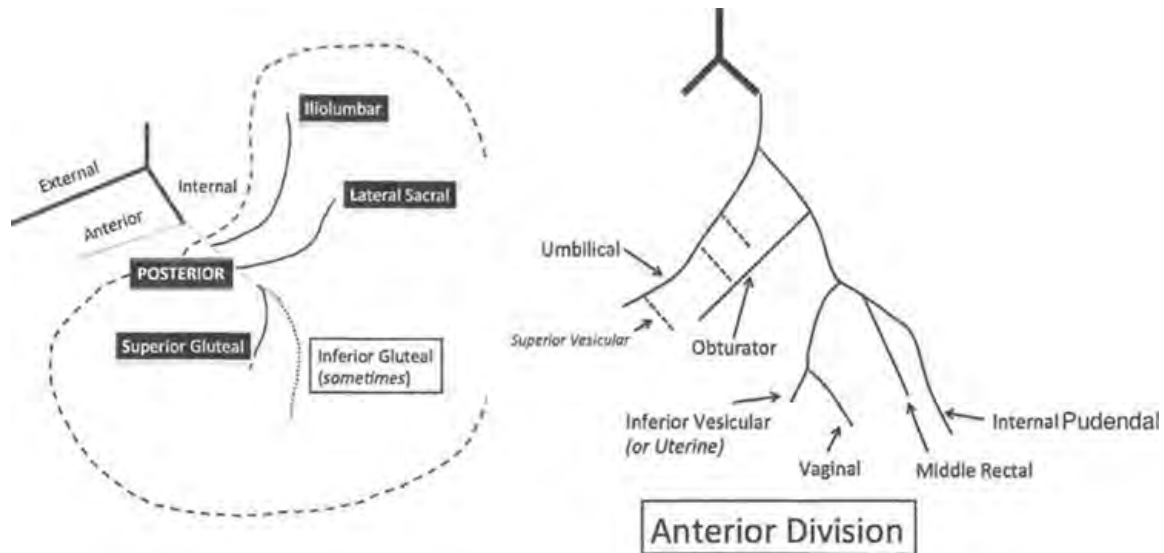
Here is the big point (likely question): **The proper right hepatic artery is anterior to the right portal vein, whereas the replaced right hepatic artery is posterior to the main portal vein.** This positioning of the replaced right increases the risk of injury in pancreatic surgeries.

Accessory Right Inferior Hepatic Vein - This is the most common hepatic venous variant (drains segment 6 and 7 into the IVC). It only matters for pre-op transplant stuff.

Normal Celiac Axis Anatomy:



Iliac Anatomy: The branches of the internal iliac are high yield, with the most likely question being “which branches are from the posterior or anterior divisions?” A *useful mnemonic is “I Love Sex, ” Illiolumbar, Lateral Sacral, Superior Gluteal, for the posterior division*. I don’t think they will actually show a picture, it’s way more likely to be a written question.



Lower Extremity Anatomy - The highest yield thing to know is that the **anterior tibialis is the first branch off the popliteal**. The second highest yield thing to know is when stuff becomes what.

- * *Common Femoral Artery (CFA): Begins at the level of inguinal ligament*
- * *Superficial Femoral Artery (SFA): Begins once the CFA gives off the profunda femoris*
- * *Popliteal Artery: Begins as the SFA exits the adductor canal*
- * *Popliteal Artery terminates as the anterior tibial artery and the tibioperoneal trunk*

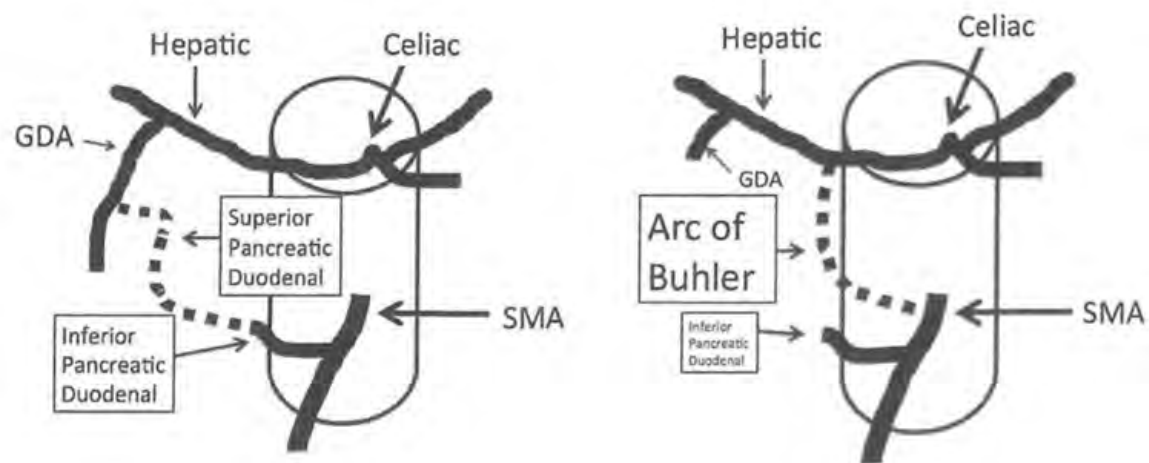
Upper Extremity Anatomy - The highest yield thing you can know is when stuff becomes what: *

- * *Axillary Artery: Begins at the first rib*
- * *Brachial Artery: Begins as it crosses the teres major*
- * *Brachial Artery: Bifurcates to the ulnar and radial artery*
- * *Intraosseous Branch: Typically arises from the ulnar*
- * *Superficial Arch = Arises from the Ulna*
- * *Deep Arch = Arises from the Radius*

Arterial Collateral Pathways:

Celiac to SMA: The conventional collateral pathway is Celiac -> Inferior Pancreatic Duodenal -> Superior Pancreatic Duodenal -> GDA.

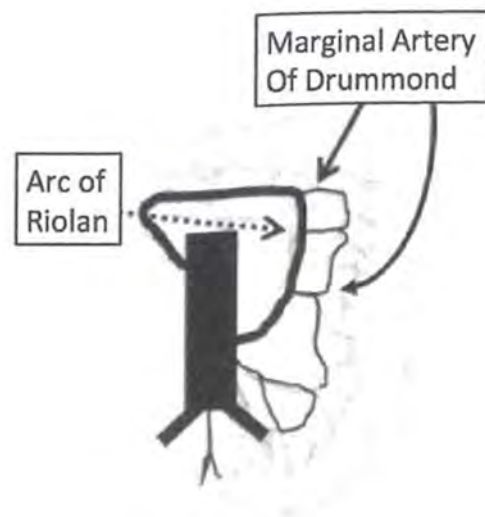
Arc of Buhler: This is a variant anatomy (seen in like 4% of people), that represents a collateral pathway from the celiac to the SMA. The arch is independent of the GDA and inferior pancreatic arteries. This rare collateral can have an even more rare aneurysm, which occurs in association with stenosis of the celiac axis.



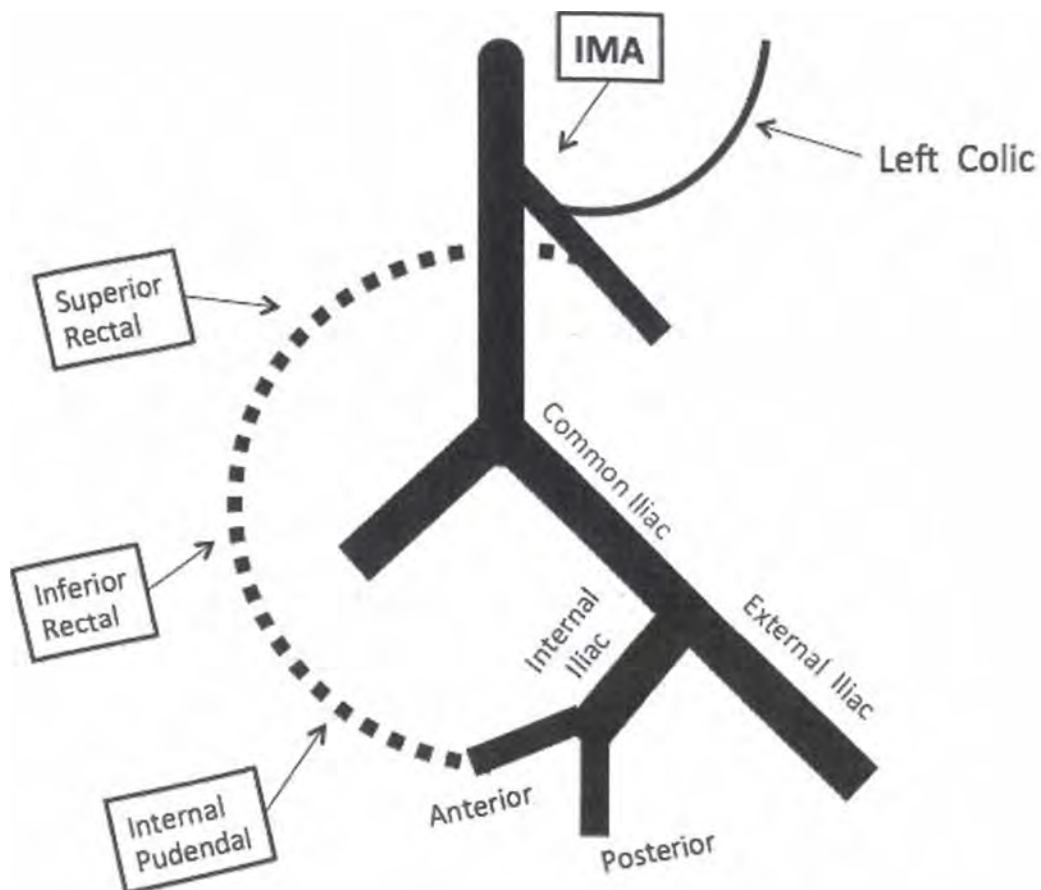
SMA to IMA: The conventional collateral pathway is SMA -> Middle Colic -> Left Branch of the Middle Colic -> Arc of Riolan (as below) -> Left Colic -> IMA.

Arc of Riolan - Also referred to as the meandering mesenteric artery. Classically a **connection between the middle colic of the SMA and the left colic of the IMA.**

Marginal Artery of Drummond - This is another **SMA to IMA connection.** The anastomosis of the terminal branches of the ileocolic, right colic and middle colic arteries of the SMA, and of the left colic and sigmoid branches of the IMA, form a **continuous arterial circle or arcade along the inner border of the colon.**

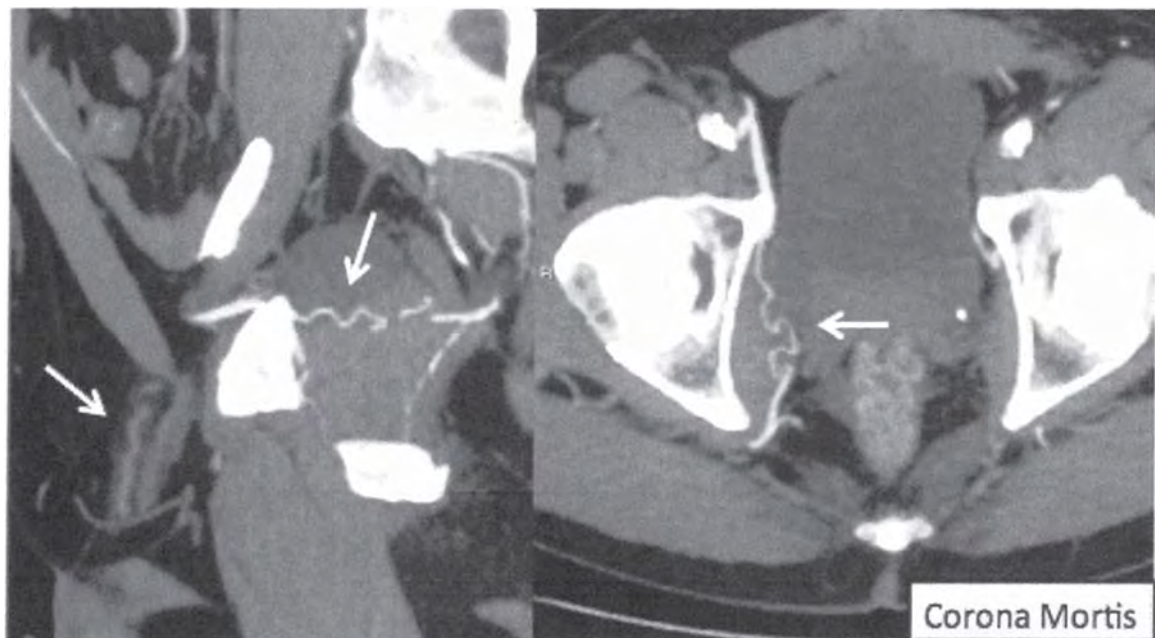


IMA to Iliacs: - The conventional collateral pathway is IMA -> Superior Rectal -> Inferior Rectal -> Internal Pudendal -> Anterior branch of internal iliac.



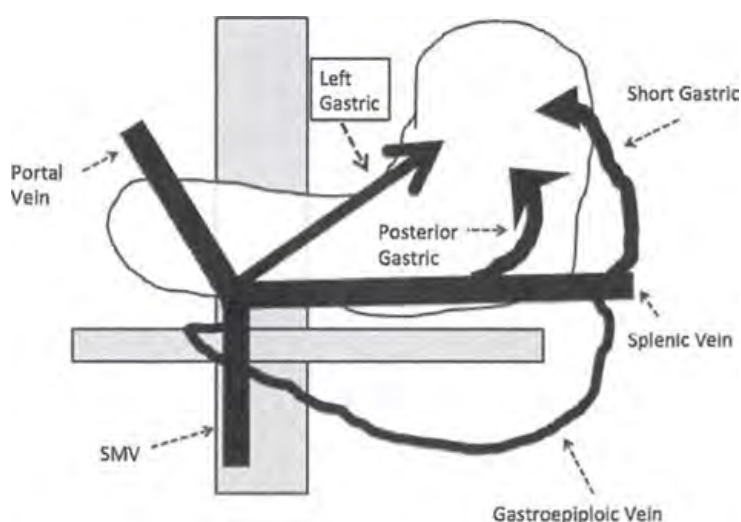
Winslow Pathway - This is a collateral pathway that is seen in the setting of aorto-iliac occlusive disease. The pathway apparently can be inadvertently cut during transverse abdominal surgery. The pathway runs from subclavian arteries -> internal thoracic (mammary) arteries -> superior epigastric arteries -> inferior epigastric arteries -> external iliac arteries.

Corona Mortis - Classically described as a vascular connection between the **obturator and external iliac**. Some authors describe additional anastomotic pathways, but you should basically think of it as any vessel **coursing over the superior pubic rim**, regardless of the anastomotic connection. The “crown of death” is significant because it can (a) be **injured in pelvic trauma** or (b) be **injured during surgery - and is notoriously difficult to ligate**. Some authors report that it causes 6-8% of deaths in pelvic trauma. The last piece of trivia is that it could hypothetically cause a type 2 endoleak.



Venous Collaterals:

Gastric Varices: - As described in more detail in the GI chapter, portal hypertension shunts blood away from the liver and into the systemic venous system. Spontaneous portal-systemic collaterals develop to decompress the system. The things to know are that **most gastric varices are formed by the left gastric (coronary vein)**. This is the one they always show big and dilated on an angiogram. Isolated gastric varices are secondary to splenic vein thrombosis. Gastric Varices (80-85%) drain into the inferior phrenic and then into the left renal vein, forming a gastro-renal shunt.



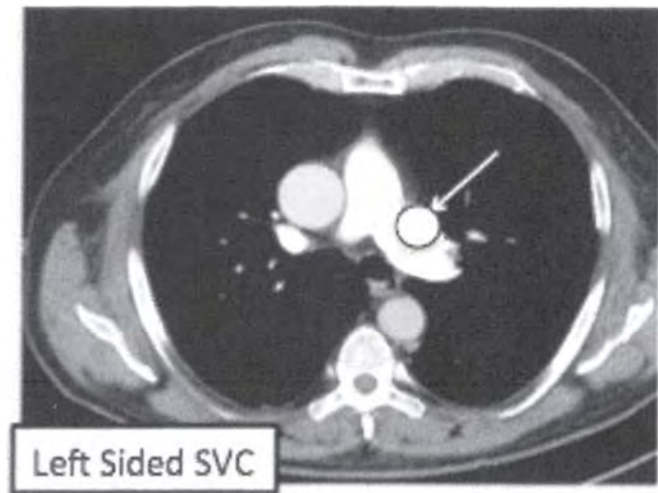
If anyone asks:

- Left Gastric (Coronary) = Cardia
- Posterior and Short Gastric = Fundus

Splenorenal Shunt - Another feature of portal hypertension, this is an abnormal collateral between the splenic vein and renal vein. This is actually a desirable shunt because it is **not associated with GI bleeding**. However, **enlarged shunts are associated with hepatic encephalopathy** (discussed in greater detail in the BRTO section of the IR chapter). A common way to show this is an enlarged left renal vein and dilatation of the inferior vena cava at the level of the left renal vein.

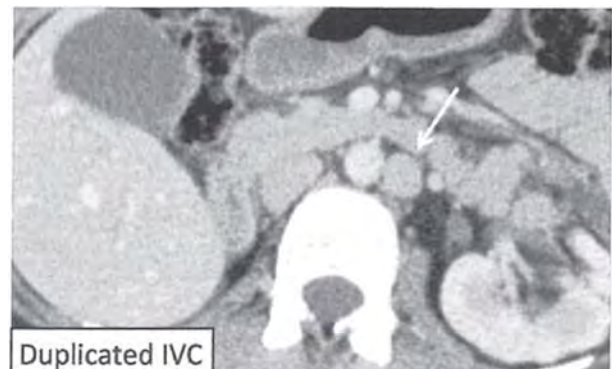
Caval Variants:

Left Sided SVC - The most common congenital venous anomaly in the chest. In a few rare cases these can actually result in a right to left shunt. They are only seen in isolation in 10% of cases (usually it's a duplicated SVC). As a piece of trivia the most common associated CHD is an ASD. Almost always (92%) of the time, a left sided SVC drains into the coronary sinus (the other 8% drain into the left atrium). The location and appearance is a total **Aunt Minnie**.



Duplicated SVC - This is usually seen in the scenario of a left sided SVC, with a smaller right SVC also present.

Duplicated IVC - There are two main points worth knowing about this: (1) that the appearance is an **Aunt Minnie**, and (2) it's **associated with Renal stuff**. Renal associations include horse shoe and crossed fused ectopic kidneys. Also these dudes often have circumaortic renal collars (see below).

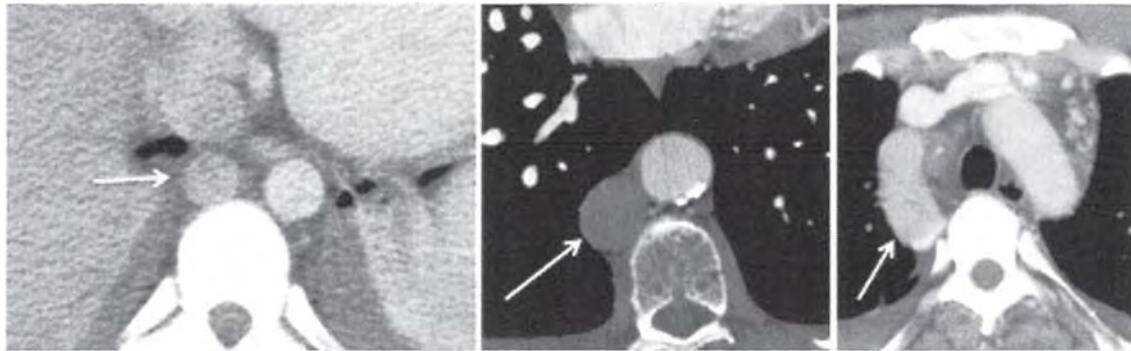


Circumaortic Venous Collar - Very common variant with an additional left renal vein that passes posterior to the aorta. It only matters in two situations (a) renal transplant, (b) IVC filter placement. The classic question is that the **anterior limb is superior**, and the posterior limb is inferior.



Azygos Continuation - This is also known as absence of the hepatic segment of the IVC. In this case, the hepatic veins drain directly into the right atrium. Often the IVC is duplicated in these patients, with the left IVC terminating in the left renal vein, which then crosses over to join the right IVC.

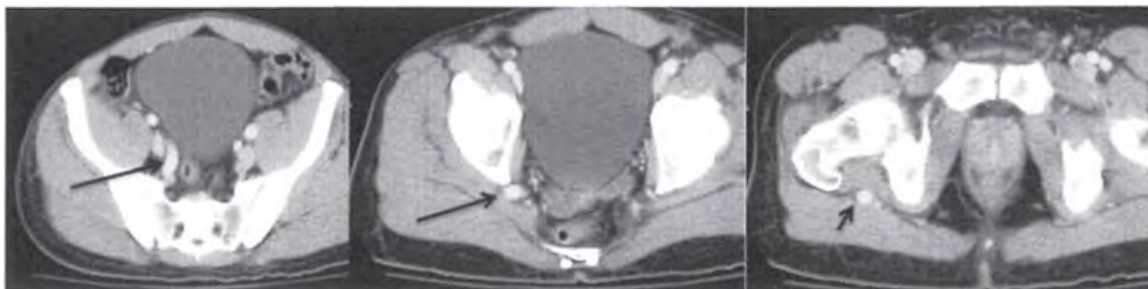
The first thing you should think when I say azygous continuation is **polysplenia** (reversed IVC/Aorta is more with asplenia).



Azygos Continuation - No IVC in the Liver, Dilated Azygos in the chest

Other Variants:

Persistent Sciatic Artery-An anatomic variant, which is a **continuation of the internal iliac**. It passes posterior to the femur in the thigh and then will anastomose with the distal vasculature. Complications worth knowing include aneurysm formation and early atherosclerosis in the vessel. The classic vascular surgery boards question is “external iliac is acutely occluded, but there is still a strong pulse in the foot”, the answer is the patient has a persistent sciatic.



Persistent Sciatic Artery

Pathology

Acute Aortic Syndromes - There are 3 “acute aortic syndromes”, aortic dissection, intramural hematoma, and penetrating ulcer.

Aortic Dissection: This is the most common cause of acute aortic syndrome (70%), and is most commonly **caused by hypertension (70%)**. This can be described as either acute (< 2 weeks), or chronic.

It can additionally be classified by location:

- * Stanford A: account for 75% of dissection and involve the ascending aorta and arch proximal to the take off of the left subclavian. These guys need to be treated surgically.
- * Stanford B: occur distal to the take off of the left subclavian and are treated medically unless there are complications (organ ischemia etc...)

Causes: Hypertension is the most frequent predisposing factor for aortic dissection.

Other associations include Marfans, Turners (Aortic valve defects), infection, and pregnancy. Cocaine has also been associated in otherwise normotensive patients.

Findings: Displacement of intimal calcifications on Non-contrast. An intimal flap is seen in only 70% of cases. When you do see two lumens, these will spiral around each other. Thrombus is located in the false lumen (which will enhance later and is generally larger than the true lumen).

True vs False Lumen:

True	False
Continuity with undissected portion of aorta	“CobWeb Sign” - slender linear areas of low attenuation
Smaller cross sectional areas (with higher velocity blood)	Larger cross section area (slower more turbulent flow)
Surrounded by calcifications (if present)	Beak Sign -
Usually contains the origin of celiac trunk, SMA, and right renal artery	Usually contains the origin of left renal artery
	Surrounds true lumen in Type A Dissection

Intimo-intimal intussusception - Unusual type of dissection. It is produced by circumferential dissection of the intimal layer, which subsequently invaginates (this has been compared to a windsock). The intimal tear usually starts near the coronary orifices.

Floating Viscera Sign: This is a classic angiographic sign of abdominal aortic dissection. It is shown as opacification of abdominal aortic branch vessels during aortography (catheter placed in the aortic true lumen), with the branch vessels—(celiac axis, superior mesenteric artery, and renal arteries) arising out of nowhere. *They appear to be floating*, with little or no antegrade opacification of the aortic true lumen

***Aneurysm with Thrombus* vs *Dissection with Thrombus in False Lumen*:**

Dissection has spiral shape, Thrombus tend to be circumferential

Mural thrombus has an irregular border, Dissection has a smooth border

Intimal Calcification displacement - favors thrombus in dissection

Intramural Hematoma: Classically seen in old hypertensives (same as dissection).

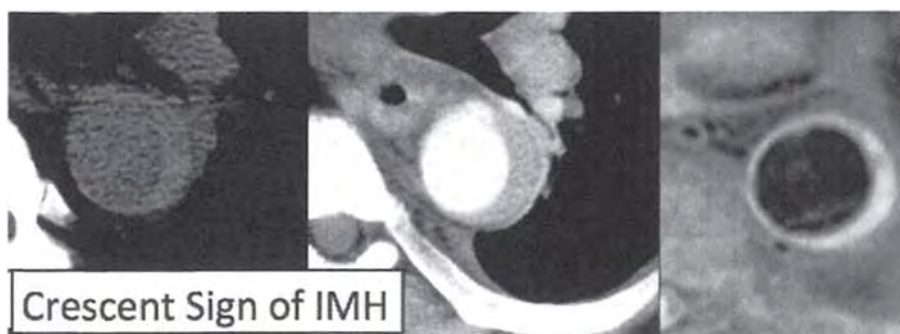
Spontaneous hemorrhage caused by rupture of the vaso vasorum in the media without an intimal tear. This can be really difficult to distinguish from a thrombosed dissection (it won't spiral the way dissection does). Can proceed to classic dissection.

Trivia:

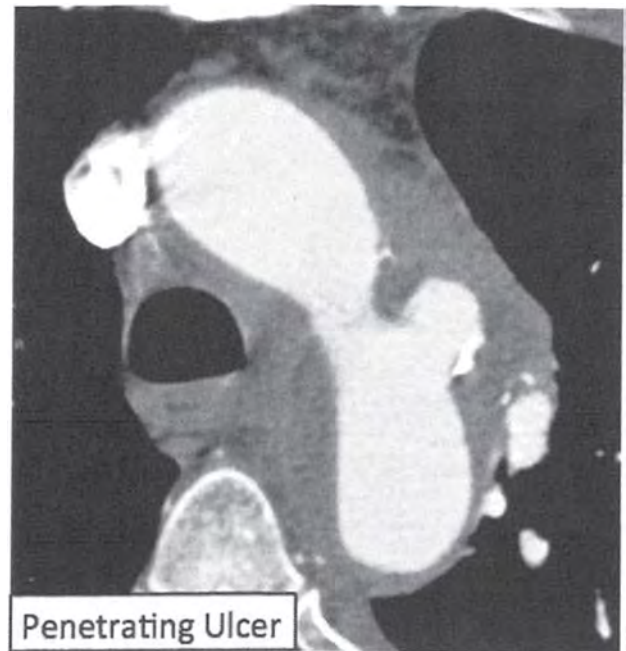
- * It's still classified Type A or Type B
- * Mortality Predictors: Ascending Aorta > 5cm, IMH > 2cm, Pericardial Effusion.

Maximum aortic diameter > 5cm is the strongest predictor to dissection.

Unenhanced CT scan depicts **crescent-shaped areas with high attenuation**. Intimal calcifications may be displaced. MRI will show T2 bright blood in the acute phase, and T1 and T2 bright blood when subacute.



Penetrating Ulcer: This is an ulceration of an atheromatous plaque that has eroded the inner elastic layer of the aortic wall. When it reaches the media it produces a hematoma within the media. These things most often occur in old people with severe underlying atherosclerosis. They can lead to saccular aneurysm. They are often multiple and therefore difficult to treat. You still use the A & B Stanford classification based on location, with corresponding medical and surgical treatment. Apparently when these guys need surgery for type Bs (symptoms etc...) they do way worse than dissected B's because they actually need surgery and can't get an endograft.



What is the highest yield testable trivia regarding Acute Aortic Syndromes?

- *Causes?*
 - Dissection & IMH = Hypertension
 - Penetrating Ulcers = Atherosclerosis
- *Stanford A vs B?*
 - Locations before of after left subclavian takeoff.
 - A is surgical, B is medical management.
- *IMH progression to dissection?*
 - IMH maximum diameter of 5cm is the strongest predictor for dissection
- *True Lumen vs False Lumen Dissection?*
 - If you aren't sure, the bigger one is probably the false one.

Other Pathology:

Aneurysm vs Pseudo-aneurysm - The distinction between a true and false aneurysm lends itself well to multiple choice testing. A true aneurysm is an enlargement of the lumen of the vessel to 1.5 times its normal diameter. **True = 3 layers are intact.** In a **false (pseudo) aneurysm all 3 layers are NOT intact**, and it is essentially a contained rupture. The risk of actual rupture is obviously higher with false aneurysm. It can sometimes be difficult to tell, but as a general rule fusiform aneurysms are true, and saccular aneurysms might be false. Classic causes of pseudoaneurysm include trauma, cardiologists (groin sticks), infection (mycotic), pancreatitis, and some vasculitides. On ultrasound they could show you the classic ying/yang sign, with “to and fro” flow on pulsed Doppler. The yin/yang sign can be seen in saccular true aneurysms, so you shouldn’t call it on that alone (unless that’s all they give you). **To and Fro flow within the aneurysm neck + clinical history is the best way to tell them apart.**

SVC Syndrome - Occurs secondary to complete or near complete obstruction of flow in the SVC from external compression (lymphoma, lung cancer) or intravascular obstruction (Central venous catheter, or pacemaker wire with thrombus). A less common but testable cause is fibrosing mediastinitis (just think histoplasmosis). The dude is gonna have face, neck, and bilateral arm swelling.

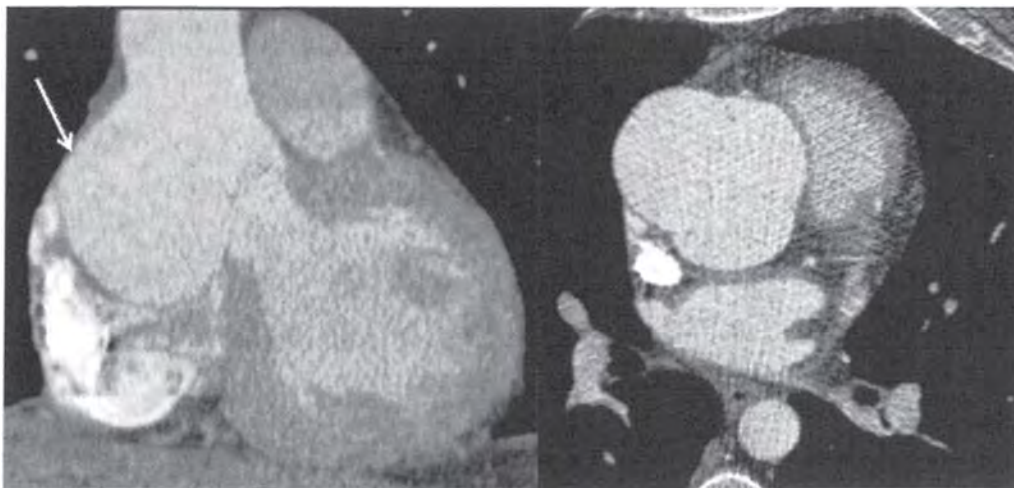
Traumatic Pseudoaneurysm - Again a pseudoaneurysm is basically a contained rupture. The most common place to see this (in a living patient) is the **aortic isthmus (90%)**. This is supposedly the result of tethering from the ligamentum arteriosum. The second and third most common sites are the ascending aorta and diaphragmatic hiatus. Ascending aortic injury is actually probably number one, it just kills them in the field so you don’t see it. They could show you a CXR with a wide mediastinum, deviation of the NG Tube to the right, depressed left main bronchus, or left apical cap and want you to suspect acute injury.



Ascending Aortic Calcifications - There are only a few causes of ascending aortic calcifications, as atherosclerosis typically spares the ascending aorta. Takayasu and Syphilis should come to mind. The real life significance is the clamping of the aorta may be difficult during CABG.

Aneurysm - Defined as enlargement of the artery to 1.5 times its expected diameter (> 4cm of the Ascending and Transverse, > 3.5cm Descending, > 3.0 cm Abdominal). Atherosclerosis is the most common overall cause. Medial degeneration is the most common cause in the ascending aorta. Patients with connective tissue (Marfans, Ehlers Danlos) diseases tend to involve the aortic root. *When I say cystic medial necrosis you should think Marfans.* Aneurysms may develop in any segment of the aorta, but most involve the infra-renal abdominal aorta. This varies based on risk factors, rate of growth, etc... but a general rule is surgical repair for aneurysms at 6cm in the chest (5.5cm with collagen vascular disease) and 5cm in the abdomen.

Sinus of Valsalva Aneurysm - Aneurysms of the valsalva sinus (aortic sinus) are rare in real life, but have been known to show up on multiple choice tests. Factoids worth knowing are that they are more common in Asian Men, and typically involve the right sinus. They can be congenital or acquired (infectious). VSD is the most common associated cardiac anomaly. Rupture can lead to cardiac tamponade. Surgical repair with Bentall procedure.



Sinus of Valsalva Aneurysm

Endoleaks: - There are 5 types, and type 2 is the most common. These are discussed in detail in the IR chapter.

Rupture /Impending Rupture- A retroperitoneal hematoma adjacent to an AAA is the most common imaging finding of rupture. The most common indicator for elective repair is the maximum diameter of the aneurysm (“Sac Size Matters”). A thick circumferential thrombus is thought to be protective against rupture. Enlargement of the patent lumen can indicate lysis of thrombus and predispose to rupture.

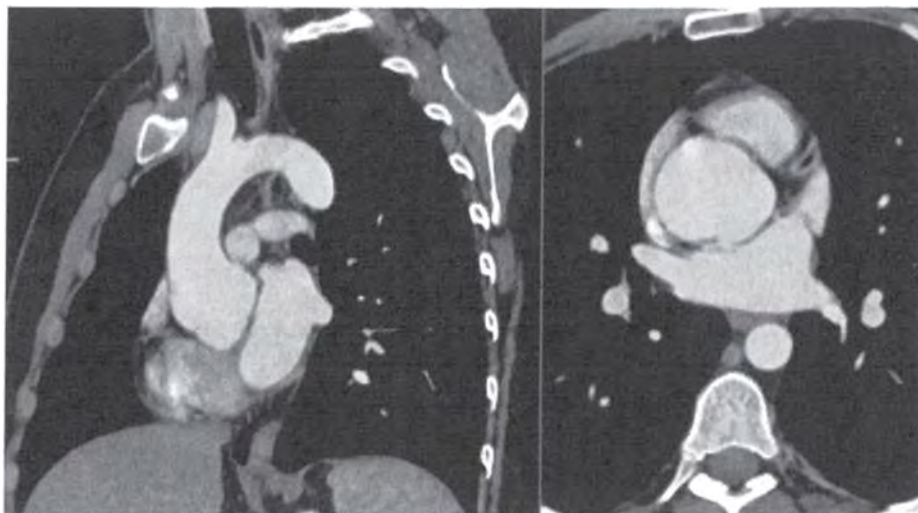
Findings of Impending Rupture	
Draped Aorta Sign	Posterior wall of the aorta drapes over the vertebral column.
Increased Aneurysm Size	10mm or more increased per year
Focal Discontinuity in Circumferential Wall Calcifications	
Hyperdense Crescent Sign	Well defined peripheral crescent of increased attenuation. One of the most specific manifestations of impending rupture.

Mycotic Aneurysm - These are most often **saccular** and most often **pseudoaneurysms**. They are prone to rupture. They most often occur via hematogenous seeding in the setting of septicemia (**endocarditis**). They can occur from direct seeding via a psoas abscess or vertebral osteomyelitis (but this is less common). **Most occur in the thoracic or supra-renal aorta** (most atherosclerotic aortic aneurysms are infra-renal). Typical findings include saccular shape, lobular contours, periaortic inflammation, abscess, and periaortic gas. They tend to expand faster than atherosclerotic aneurysms. In general small, asymptomatic, and unruptured infected aneurysms can get IV antibiotics. Large, ruptured, or symptomatic ones get **OPEN surgery** (don’t stent graft it - although this is controversial).

NF 1 - One of the more common neurological genetic disorders, which you usually think about causing all the skin stuff (Cafe au lait spots, and freckling), and bilateral optic gliomas. Although uncommon, vascular findings also occur in this disorder. **Aneurysms and stenosis are also sometimes seen in the aorta and larger arteries**, while dysplastic features are found in smaller vessels. **Renal artery stenosis can occur leading to renovascular hypertension** (found in 5% of children with NF). **The classic look is orificial renal artery stenosis presenting with hypertension in a teenager or child**. The mechanism is actually Dysplasia of the arterial wall itself (less common from peri-arterial neurofibroma).

Marfan Syndrome - Genetic disorder caused by mutations of the fibrillin gene (step 1 question). There are lots of systemic manifestations including ectopic lens, being tall, pectus deformity, scoliosis, long fingers etc... Vascular findings can be grouped into aneurysm, dissection, and pulmonary artery dilation:

- * *Aneurysm*: Dilation with Marfans is classically described as “**Annuloaortic ectasia**”, with dilatation of the aortic root. The dilation usually begins with the aortic sinuses, and then progresses into the sinotubular junction , ultimately involving the aortic annulus. **Dilatation of the aortic root leads to aortic valve insufficiency**. Severe aortic regurgitation occurs that may progress to aortic root dissection or rupture. The mechanism for all this nonsense is that disruption of the media elastic fibers causes aortic stiffening, and predisposes to aneurysm and dissection. The buzzword for the Marfans ascending aneurysm is “**tulip bulb.**” They are usually repaired earlier than normal aneurysm (typically around 5.5cm).
- * *Dissection*: Recurrent dissections are common, and even “triple barreled dissection” can be seen (dissections on both sides of a true channel).
- * *Pulmonary Artery Enlargement*: Just like dilation of the aorta, pulmonary artery enlargement favors the root.



Marfan's - *Annuloaortic Ectasia*", with dilatation of the aortic root.

Loeys Dietz Syndrome - Just think of this as the really shitty version of Marfans. They have a terrible prognosis, and rupture their aortas all the time. **Vessels are very tortuous** (twisty). They also have crazy wide eyes (hypertelorism).

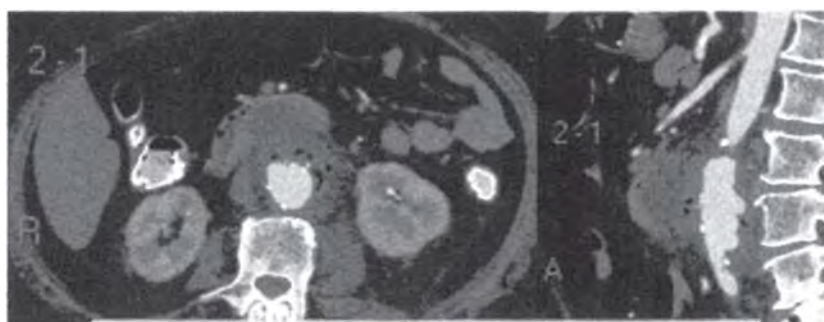
Ehlers Danlos - This one is a disorder in collagen, with lots of different subtypes. They have the stretchy skin, hypermobile joints, blood vessel fragility with bleeding diatheses. Invasive diagnostic studies such as conventional angiography and **other percutaneous procedures should be avoided** because of the excessive risk of arterial dissection. Imaging characteristics of aortic aneurysms in Ehlers-Danlos syndrome **resemble those in Marfan syndrome, often involving the aortic root**. Aneurysms of the abdominal visceral arteries are common as well.

Syphilitic (Luetic) aneurysm - This is super rare and only seen in patients with untreated tertiary syphilis. There is classically a saccular appearance and involves the ascending aorta as well as the aortic arch aorta. Classic description **“saccular asymmetric aortic aneurysm with involvement of the aortic root branches.”** Often heavily calcified **“tree bark” intimal calcifications**. Coronary artery narrowing (at the ostium) is seen 30% of the time. Aortic valve insufficiency is also common.

Aorto-Enteric Fistula - These come in two flavors: (a) Primary, and (b) Secondary.

- * **Primary:** Very, very, very rare. Refers to an A-E fistula without history of instrumentation. They are only seen in the setting of aneurysm and atherosclerosis.
- * **Secondary:** Much more common. They are **seen after surgery** with or without stent graft placement.

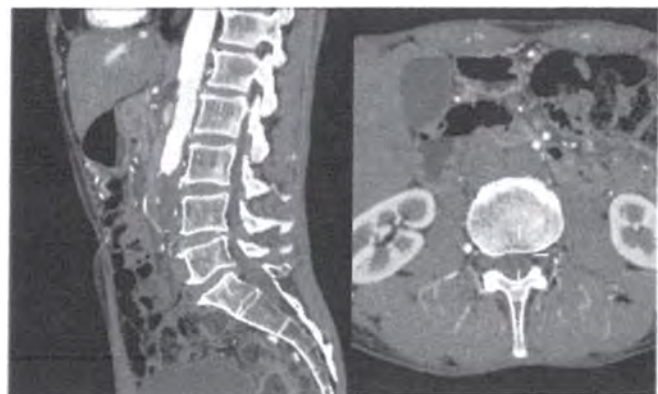
The question is usually what part of the bowel is involved, and the answer is 3rd **and 4th portions of the duodenum**. The second most likely question is A-E fistula vs perigraft infection (without fistula)? The answer to that is unless you see contrast from the aorta into the bowel lumen (usually duodenum), you can't tell. Both of them have ectopic perigraft gas > 4 weeks post repair, both have perigraft fluid and edema, both lose the fat plane between the bowel and aorta (tethering of the duodenum to the anterior wall of the aorta), both can have pseudoaneurysm formation.



Aorto-Enteric Fistula - *Primary Type*

Inflammatory Aneurysms - Most are **symptomatic**, more common in **young men**, and associated with increased risk of rupture regardless of their size. Unlike patients with atherosclerotic AAA, most with the inflammatory variant have an **elevated ESR**. Their etiology is not well understood but may be related to periaortic retroperitoneal fibrosis or other autoimmune disorders (SLE, Giant Cell, RA). **Smoking is apparently a strong risk factor**, and smoking cessation is the first step in medical therapy. **In 1/3 of cases hydronephrosis or renal failure** is present at the time of diagnosis because the inflammatory process usually involves the ureters. Imaging findings include a thickened wall, inflammatory or fibrotic changes in the periaortic regions. Often there is asymmetrical thickening of the aorta with sparing of the posterior wall (helps differentiate it from vasculitis).

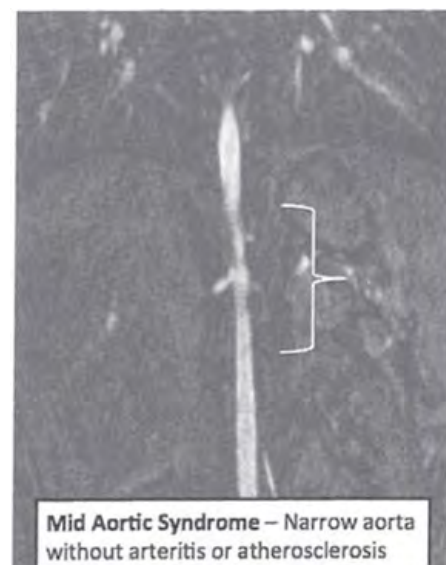
Leriche Syndrome - Refers to complete occlusion of the aorta distal to the renal arteries (most often at the aortic bifurcation). It is often secondary to bad atherosclerosis. There can be large collaterals.



Leriche Syndrome - Complete occlusion of the aorta distal to the renal arteries

The most likely question is the **triad: 1. Ass Claudication, 2. Absent/ Decreased femoral pulses, 3. Impotence.**

Mid Aortic Syndrome: - Refers to progressive narrowing of the abdominal aorta and its major branches. Compared to Leriche, this is higher, and longer in segment. It's also a total freaking zebra. It tends to affect children / young adults. This thing is characterized by progressive narrowing of the aorta. It is **NOT secondary to arteritis or atherosclerosis** but instead the result of some intrauterine insult (maybe) with fragmentation of the elastic media.



This also has a triad: **1. HTN (most common presenting symptom), 2. Claudication, 3. Renal failure.**

Aortic Coarctation: This comes in two flavors:

- * Infantile (Pre-ductal) - these guys can have pulmonary edema. More typically a long segment. Blood supply to the descending aorta is via the PDA.
- * Adult (Ductal) - Not symptomatic until later in childhood. Often presents with differential arm/leg blood pressures. More typically a short segment.

OK, things to know: **Strong Association with Turners Syndrome (15-20%). Bicuspid Aortic valve is the most common associated defect (80%).** They have more berry aneurysms. Figure 3 sign (appearance of CXR). Rib Notching: most often involves 4th - 8th ribs. It does NOT involve the 1st and 2nd because those are fed by the costocervical trunk.

Pseudocoarctation: This is a favorite of multiple choice writers. You will have elongation with narrowing and kinking of the aorta. It really looks like a coarctation, BUT there is **NO pressure gradient, collateral formation, or rib notching** - that is the most likely question. The second most likely question is the area of aneurysmal dilation may occur distal to the areas of narrowing in pseudocoarctation, and they may become progressively dilated and should therefore be followed.

Thoracic Outlet Syndrome - Congenital or acquired compression of the Subclavian vessels (artery and vein), and brachial plexus nerves as they pass through the thoracic inlet. *It is a spectrum: Nerve (95%) »»» Subclavian Vein » Subclavian Artery.* With symptoms varying depending on what is compressed. **Compression by the anterior scalene muscle is the most common cause.** However, cervical rib, muscular hypertrophy, fibrous bands, pagets, tumor etc... can all cause symptoms. Treatment is usually surgical removal of the rib / muscle. The way they will show this is arms up and arms down angiography (occlusion occurs with arms up).

Paget Schroetter - This is **essentially thoracic outlet syndrome, with development of a venous thrombus in the Subclavian vein.** It's sometimes called "effort thrombosis" because it's associated with athletes (pitchers, weightlifters) who are raising their arms a lot. They will use catheter directed lysis on these dudes, and surgical release of the offending agent as above. Stenting isn't usually done (and can only be done after surgery to avoid getting the stent crushed).

Pulmonary Artery Aneurysm/Pseudoaneurysm - Think about three things for multiple choice;

(1) **Iatrogenic from swan ganz catheter** ***most common** (2) Behcets, (3) Chronic PE.

When they want to lead swan ganz they may say something like “patient in the ICU.” The buzzwords for Behcets are: “Turkish descent”, and “mouth and genital ulcers.”

- * **Hughes-Stovin Syndrome:** This is a zebra cause of pulmonary artery aneurysm that is similar (and maybe the same thing) as Behcets. It is characterized by recurrent thrombophlebitis and pulmonary artery aneurysm formation and rupture.
- * **Rasmussen Aneurysm:** This has a cool name, which instantly makes it high yield for testing. This is a pulmonary artery **pseudoaneurysm secondary to pulmonary TB**. It usually involves the upper lobes in the setting of reactivation TB.
- * **Tetrolgy of Fallot Repair Gone South:** So another possible testable scenario is the patch aneurysm, from the RYOT repair.

Mesenteric Ischemia: - This can be broadly classified as acute or chronic.

Chronic: Significant Stenosis of 2 out of 3 main mesenteric vessels + symptoms (“food fear”), LUQ pain after eating, pain out of proportion to exam). Some practical pearls are that you can have bad disease and no symptoms if you have good collaterals. Alternatively if you have bad one-vessel disease you can have symptoms if you have crappy collaterals.

Remember that the *splenic flexure is the most common* because it’s the watershed of the SMA and IMA.

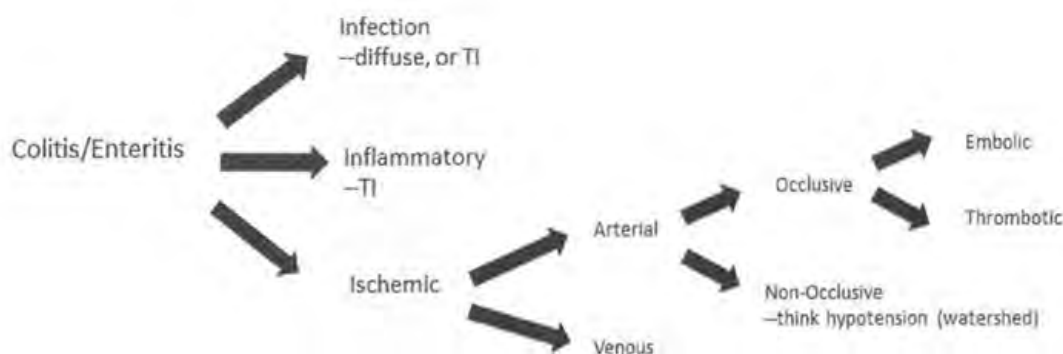
Acute: This comes from 4 main causes. Arterial, Venous, Hypovolemic, and Strangulation.

- * **Arterial:** Occlusive emboli (usually more distal, at branch points), or Thrombus (usually closer to the ostium). Vasculitis can also cause it. The SMA is most commonly affected. Arterial typically has a **thinner wall** (no arterial inflow), and is **NOT typically dilated**. After reperfusion the bowel wall will become thick, with target appearance.
- * **Venous: Dilation with wall thickening** (8-9mm, with < 5mm being normal) is more common. Fat stranding and ascites are especially common findings in venous occlusion.
- * **NonOcclusive:** Seen in patients in shock or on pressors. This is the most difficult to diagnose on CT. The involved segments are often thickened. Enhancement is variable. *Look for delayed fdling of the portal vein at 70 seconds.*

Strangulation: This is almost always secondary to a closed loop obstruction. This is basically a mixed arterial and venous picture, with **congested dilated bowel**. Hemorrhage may be seen in the bowel wall. The lumen is often fluid filled.

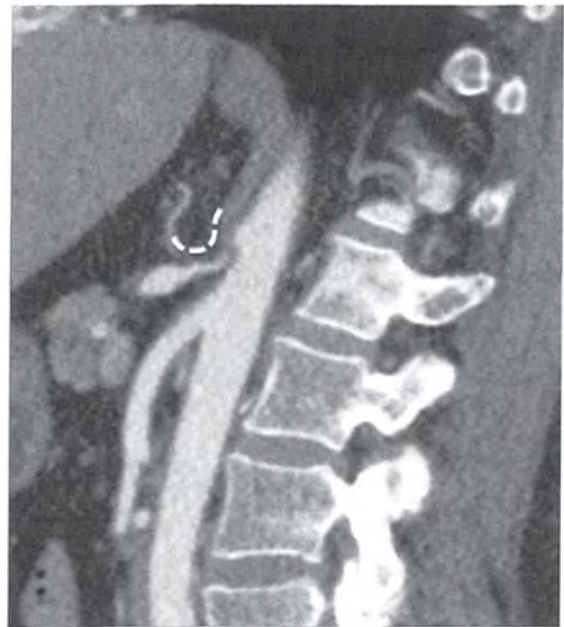
Mesenteric Ischemia			
Arterial	Venous	Strangulation	Nonocclusive
Thin Bowel Wall (thick after reperfusion)	Thick Bowel Wall	Thick Bowel Wall	Thick Bowel Wall
Diminished Enhancement	Variable	Variable	Variable
Bowel Not Dilated	Moderate Dilation	Severe Dilation (and fluid filled)	Bowel Not Dilated
Mesentery Not Hazy (until it infarcts)	Hazy with Ascites	Hazy with Ascites, and “whirl sign” with closed loop.	Mesentery Not Hazy (until it infarcts)

This is my general algorithm if I see angry bowel:



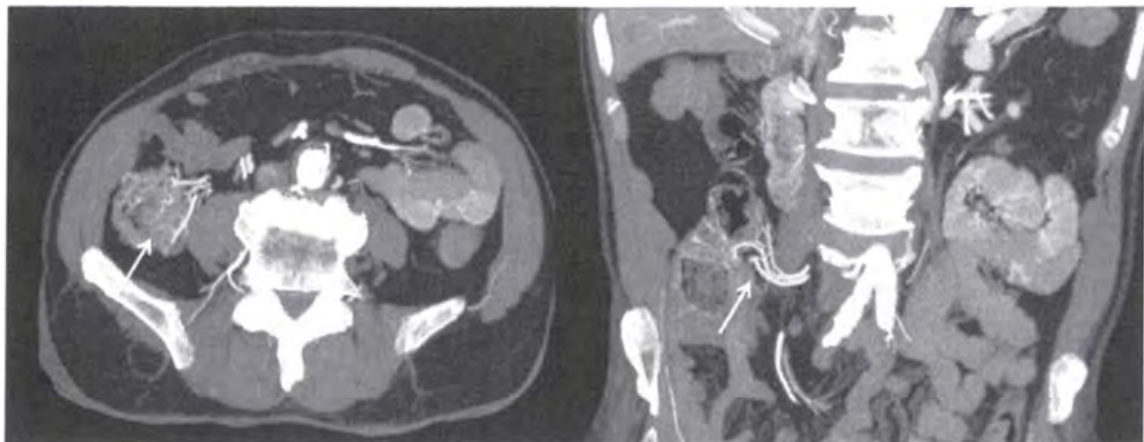
Splenic Artery Aneurysm: The most common visceral arterial aneurysm. They can be true or false. The true ones are associated with HTN, portal HTN, cirrhosis, liver transplant, and **pregnancy. More common in pregnancy, and more likely to rupture in pregnancy.** In contrast to normal aneurysms **atherosclerosis is NOT considered the underlying cause.** Most are located in the distal artery. False aneurysms are associated with pancreatitis. An important mimic is the islet cell pancreatic tumor (which is hypervascular). Don't be a dumb ass and try and biopsy the aneurysm. If you are forced to choose *which ones to treat* I guess I'd go with: anything over 2cm, any false one, and anyone in a women planning on getting pregnant.

Median Arcuate Ligament Syndrome (Dunbar Syndrome) : This is compression of the celiac artery by the median arcuate ligament (fibrous band that connects the diaphragm). Most people actually have some degree of compression, but it's not a syndrome until there are symptoms (abdominal pain, weight loss). Typical age is 20-40 years old. The buzzword is **“hooked appearance.”** It's classically shown on angiography and they will want you to know that it gets **worse with expiration**. It can actually lead to the development of pancreaticoduodenal collaterals and aneurysm formation. It's treated surgically.



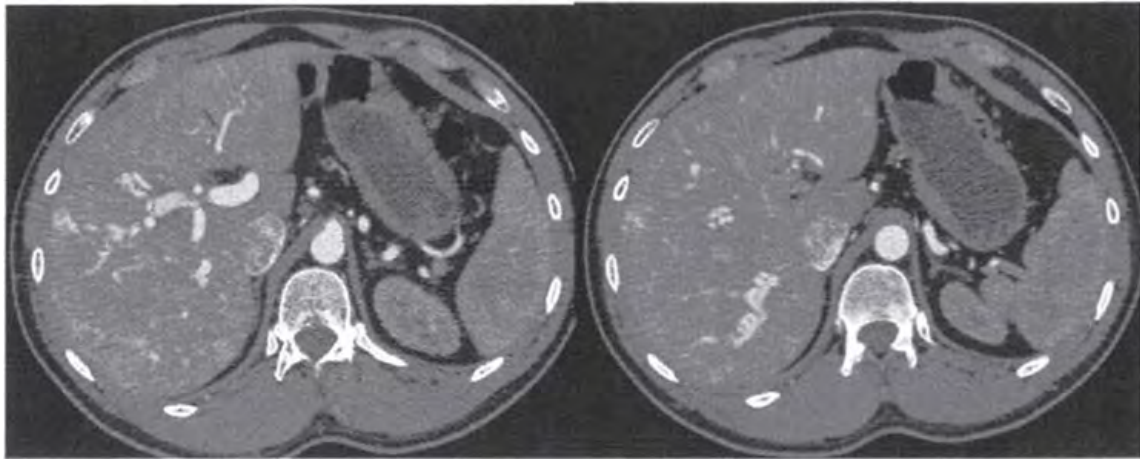
Median Arcuate Ligament

Colonic Angiodysplasia - This is the second most common cause of colonic arterial bleeding (diverticulosis being number one). This is primarily **right sided** with angiography demonstrating a cluster of small arteries during the arterial phase (along the antimesenteric border of the colon), with *early opacification of dilated draining veins* that persists late into the venous phase. There is an **association with aortic stenosis which carries the eponym Heyde Syndrome** (which instantly makes it high yield for multiple choice).



Colonic Angiodysplasia

Osier Weber Rendu (Hereditary Hemorrhagic Telangiectasia) - This is an AD multi-system disorder characterized by multiple AVMs. On step 1 they used to show you the tongue / mouth with the telangiectasis and a history of recurrent bloody nose. Now, they will likely show multiple hepatic AVMs or multiple pulmonary AVMs. Extensive shunting in the liver can actually cause biliary necrosis, and bile leak. They can have high output cardiac failure. **Most die from stroke, or brain abscess.**



Osier Weber Rendu (Hereditary Hemorrhagic Telangiectasia)

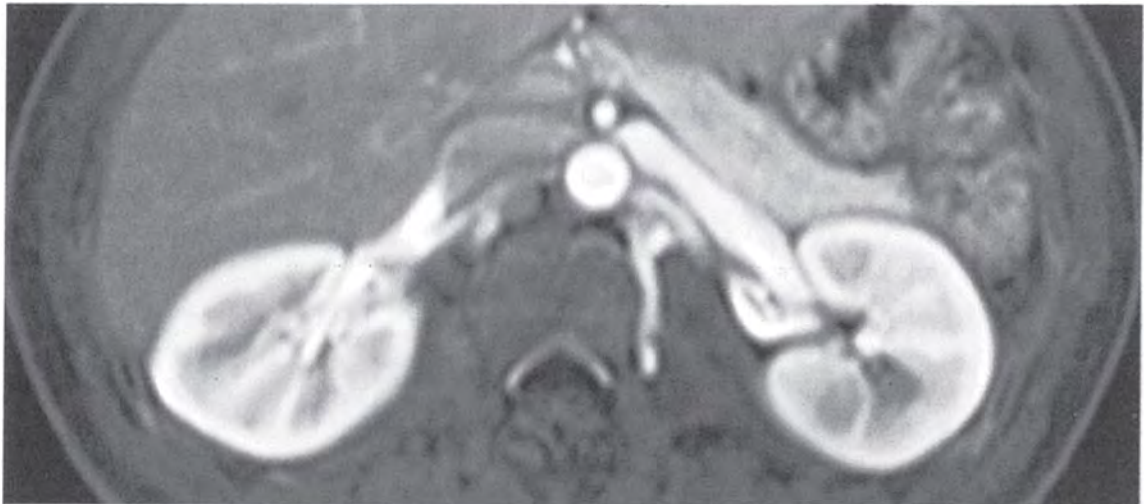
Renal Artery Stenosis - Narrowing of the renal artery most commonly occurs secondary to atherosclerosis (75%). This type of narrowing is usually near the ostium, and can be stented. FMD is the second most common cause and typically has a beaded appearance sparing the ostium (should not be stented). Additional more rare causes include PAN, Takayasu, NF-1, and Radiation.

FMD (Fibromuscular Dysplasia) - A nonatherosclerotic vascular disease, primarily affecting the renal arteries of young white women.

Things to know:

- Renovascular HTN in Young Women = FMD
- Renal arteries are the most commonly involved (carotid #2, iliac #3)
- There are 3 types, but just remember medial is the most common (95%)
- They are predisposed to spontaneous dissection
- Buzzword = String of Beads
- Treatment = Angioplasty WITHOUT stenting.

Nutcracker Syndrome - Smashing of the left renal vein as it slides under the SMA, with resulting abdominal pain(left flank) and hematuria. The left renal vein gets smashed a lot, but it's not a syndrome without symptoms. Since the left gonadal vein drains into the left renal vein, it can also cause left testicle pain in men, and LLQ pain in women.



Nutcracker: Renal Vein, smashed by SMA.
Note the prominent venous collateral

Pelvic Congestion Syndrome - This is a controversial entity, sometimes grouped in the fibromyalgia spectrum. Patients often have “chronic abdominal pain.” They also often wear a lot of rings and drink orange soda. The classic demographic is a depressed, multiparous, pre-menopausal women with chronic pelvic pain. Venous obstruction at the left renal vein (nutcracker compression) or incompetent ovarian vein valves leads to **multiple dilated parauterine veins**. This of course can be treated by your local interventional Radiologist via ovarian vein embolization.

Testicular Varicocele - Abnormal dilation of veins in the pampiniform plexus. Most cases are idiopathic and **most (98%) are found on the left side** (*left vein is longer, and drains into renal vein at right angle*). They can also occur on the left, secondary to the above mentioned “nutcracker syndrome.” They **can cause infertility**. “Non-decompressible” is a buzzword for badness. Some sources state that neoplasm is actually the most likely cause of **non-decompressible varicocele** in men over 40 years of age; (left renal malignancy invading the renal vein). Right sided varicocele can be a sign of malignancy as well. When it’s new, and on the right side (in an adult), you should raise concern for a pelvic or abdominal malignancy. New right sided varicocele in an adult should make you think renal cell carcinoma, retroperitoneal fibrosis, or adhesions. **Non-decompressible Bad, Right Bad, Left Ok, Bilateral Ok (probably).**

Uterine A VM- This can present with life threatening massive genital bleeding. Rarely they can present with CHF. They come in two flavors (a) Congenital, and (b) Acquired. **Acquired occurs after D&C**, abortion, or multiple pregnancies. They are most likely to show this on color Doppler with serpentine structures in the myometrium with low resistance high velocity patterns. This one needs embolization. Could look similar to retained products of conception (clinical history will be different, and *RPOC is usually centered in the endometrium rather than the myometrium*).

May Thurner - A syndrome resulting in DVT of the left common iliac vein. The pathology is **compression of the left common iliac vein by the right iliac artery**. Treatment is thrombolysis and stenting. *If they show you a swollen left leg, this is probably the answer.*

Popliteal Aneurysm - This is the most common peripheral arterial aneurysm (2nd most common overall, to the aorta). The main issue with these things is distal thromboembolism, which can be limb threatening. There is a strong and frequently tested association with AAA.

- * 30-50% of patients with popliteal aneurysms have a AAA
- * 10% of patients with AAA have popliteal aneurysms
- * 50-70% of popliteal aneurysms are bilateral

The **most dreaded complication of a popliteal artery aneurysm is an acute limb** from thrombosis and distal embolization of thrombus pooling in the aneurysm.

Popliteal Entrapment - Symptomatic compression or occlusion of the popliteal artery due to the developmental relationship with the **medial head of the gastrocnemius** (less commonly the popliteus). Medial deviation of the popliteal artery is supposedly diagnostic. This usually occurs in young men (<30). These patients may have *normal pulses that decrease with plantar flexion or dorsiflexion of the foot*. They will show you either a MRA or conventional angiogram in rest and then stress (dorsi / plantar flexion) to show the artery occlude.

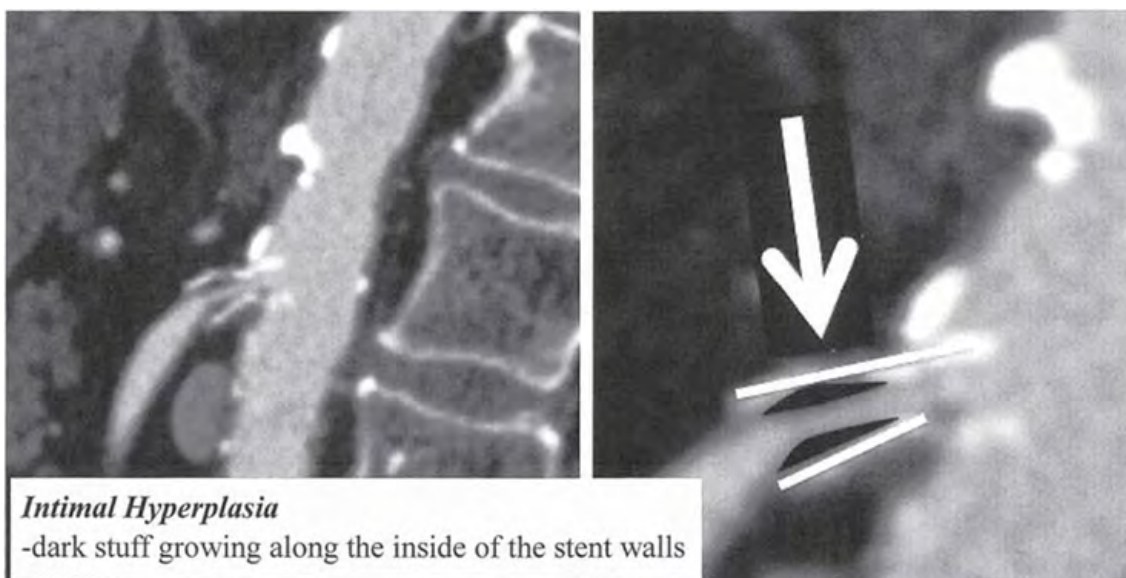
Hypothenar Hammer - Caused by blunt trauma (history of working with a jack-hammer), to the ulnar artery and superficial palmar arch. The impact occurs against the hook of the hamate. Arterial wall damage leads to aneurysm formation with or without thrombosis of the vessel. Emboli may form, causing distal obstruction of digits (this can cause confusion with the main Ddx Buerger's). Look for **corkscrew configuration of the superficial palmar arch, occlusion of the ulnar artery, or pseudoaneurysm off the ulnar artery**.

Peripheral Vascular Malformations: About 40% of vascular malformations involve the extremities (the other 40% are head and neck, and 20% is thorax). Different than hemangiomas, vascular malformations generally increase proportionally as the child grows. This dude Jackson classified vascular malformation as either low flow or high flow. Low flow would include venous, lymphatic, capillary, and mixes of the like. **High flow has an arterial component**. Treatment is basically determined by high or low flow.

Klippel-Trenaunay Syndrome (KTS) - This is often combined with **Parkes-Weber which is a true high flow AV malformation**. KTS has a triad of port wine nevi, bony or soft tissue hypertrophy (localized gigantism), and a venous malformation. A persistent sciatic vein is often associated. The marginal vein of Servelle (some superficial vein in the lateral calf and thigh) is pathognomonic (it's basically a great saphenous on the wrong side). Additional trivia: 20% have GI involvement and can bleed, if the system is big enough it can eat your platelets (**Kasabach Merritt**). Basically, **if you see a MRA/MRV of the leg with a bunch of superficial vessels (and no deep drainage) you should think about this thing**.

ABIs - So basic familiarity with the so called "Ankle to Brachial Index" can occasionally come in handy, with regard to peripheral arterial disease. This is basically a ratio of systolic pressure in the leg over systolic blood pressure in the arm. Diabetics can sometimes have unreliable numbers, because dense vascular calcifications won't let the vessels compress. 1.0 = normal, 0.5-0.9 = claudication, < 0.5 = rest pain.

Intimal Hyperplasia - “The bane of endovascular intervention.” This is not a true disease but a response to blood vessel wall damage. Basically this is an exuberant healing response that leads to intimal thickening which can lead to stenosis. You hear it talked about the most in 1R after they have revascularized a limb. Re-Stenosis that occurs 3-12 months after angioplasty is probably from intimal hyperplasia. It’s sneaky to treat and often resists balloon dilation, and/or reoccurs. If you put a bare stent in place it may grow through the cracks and happen anyway. If you put a covered stent in, it may still occur at the edges of the stent. The take home point is that it’s a pain in the ass, and if they show an angiogram with a stent in place, that now appears to be losing flow, this is probably the answer.



Vasculitis

Basically all vasculitis looks the same, with wall thickening, occlusions, dilations, and aneurysm formation. The trick to telling them apart is the age of the patient, the gender / race, and the vessels affected. Classically, they are broken up into large vessel, medium vessel, small vessel ANCA +, and small vessel ANCA negative.

Large:

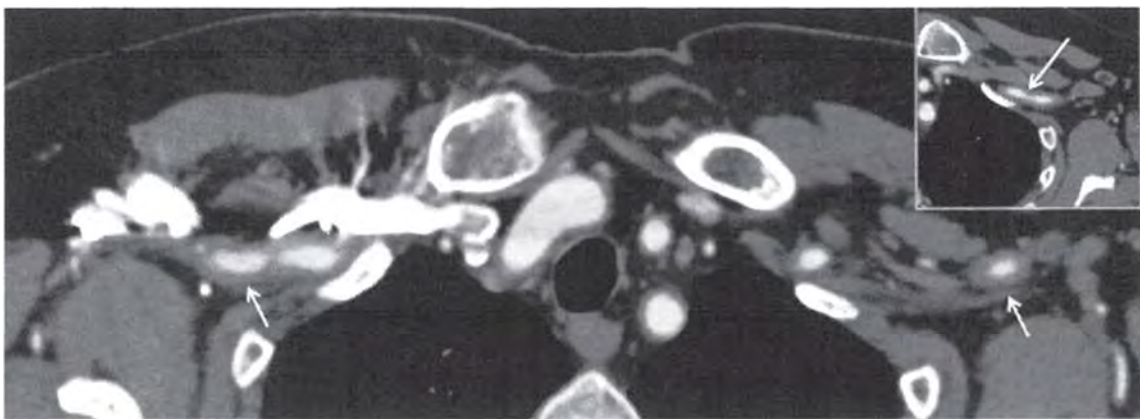
Takayasu - “The pulse-less disease.” This vasculitis loves **young Asian girls** (usually 15-30 years old). If they mention the word “Asian” this is likely to be the answer. Also, if they show you a **vasculitis involving the aorta** this is likely the answer. In the acute phase there will be both **wall thickening and wall enhancement**. There can be occlusion of the major aortic branches, or dilation of the aorta and its branches. The aortic valve is often involved (can cause stenosis or AI). In the late phase there is classically diffuse narrowing distally. The pulmonary arteries are commonly involved, with the typical appearance of peripheral pruning.

If anyone was a big enough jerk to ask, there are 6 types with variable involvement of the aorta and its branches. Which type is which is beyond the scope of the CORE, just know type 3 is most common - involves arch and abdominal aorta.



Takayasu – Wall Thickening Involving the Aorta

Giant Cell - The most common primary system vasculitis. This vasculitis loves **old men** (usually 70-80). This vasculitis involves the aorta and its major branches particularly those of the external carotid (**temporal artery**). This can be shown in two ways: (1) an ultrasound of the temporal artery, demonstrating wall thickening, or (2) CTA / MRA or even angiogram of the arm pit area (Subclavian/ Axillary/ Brachial), demonstrating wall thickening, occlusions, dilations, and aneurysm. Think about it as the **part of the body that would be compressed by crutches** (old men need crutches). Trivia worth knowing is that ESR and CRP are markedly elevated, and that the disease responds to steroids. “Gold Standard” for diagnosis is temporal artery biopsy (although it’s often negative).



Giant Cell - *"Arm Pit" Vessel Thickening*

Cogan Syndrome - Total Zebra probably not even worth mentioning. It is a large vessel vasculitis that affects children and young adults. It likes the eyes and ears causing optic neuritis, uveitis, and audiovestibular symptoms resembling Menieres. They can also get aortitis, and those that do have a worse prognosis. Basically, **kid with eye and ear symptoms + or - aortitis**.

Medium

PAN (polyarteritis nodosa) - This is one of two vasculitis (*the other being Buerger's*) that is more common in men. **PAN is more common in a MAN**. This can effect a lot of places with the big 3 being Renal (90%), Cardiac (70%), and GI (50-70%). Typically we are talking about **microaneurysm formation**, primarily at branch points, followed by infarction. I would expect this to be shown either as a CTA or angiogram of the **kidneys with microaneurysms**, or a kidney with areas of infarct (multiple wedge shaped areas). Trivia to know is the **association with Hep B**. Also, as a point of trivia the microaneurysm formation in the kidney can also be seen in patients who abuse Crystal Meth (sometimes called a “speed kidney”).

Kawasaki Disease - Probably the most common vasculitis in children (HSP also common). Think about this as a cause of coronary vessel aneurysm. A **calcified coronary artery aneurysm shown on CXR is a very rare aunt Minnie**. Other trivia includes the buzzwords “*Mucocutaneous lymph node syndrome*” and “*Fever for Five days*.”

Small Vessel Disease (ANCA +)

Wegeners - I think about upper respiratory tract (sinuses), and lower respiratory tract (lungs), and kidneys. cANCA is (+) 90% of the time. Ways this is shown are the **nasal perforation** (like a cocaine addict), and the **cavitary lung lesions**.

Churg Strauss - This is a necrotizing pulmonary vasculitis which is in the spectrum of Eosinophilic lung disease. They always have asthma and eosinophilia. **Transient peripheral lung consolidation** or ground glass regions is the most frequent feature. Cavitation is rare (this should make you think Wegeners instead). They are pANCA (+) 75% of the time.

Microscopic Polyangiitis — Affects the kidneys and lungs. Diffuse pulmonary hemorrhage is seen in about 1/3 of the cases. It is pANCA (+) 80% of the time.

Small Vessel Disease (ANCA -)

HSP (Henoch-Schonlein Purpura) - The most common vasculitis in children (usually age 4-11). Although it is a systemic disease, GI symptoms are most common (pain, blood diarrhea). It is a common lead point for intussusception. They could show this two classic ways: (1) ultrasound with a **doughnut sign for intussusception**, or (2) as a ultrasound of the **scrotum showing massive skin edema**. A less likely (but also possible) way to show this case would be multi-focal bowel wall thickening, or a plain film with thumb printing.

Behcets - Classic history is mouth ulcers, and genital ulcers, in someone with Turkish descent. It can cause thickening of the aorta, but for the purpose of multiple choice test I expect the question will be **pulmonary artery aneurysm**.

Buerger's - This vasculitis is strongly associated with **smokers**. It affects both small and medium vessels in the arms and legs (more common in legs). Although it is more commonly seen in the legs, it is more commonly tested with a hand angiogram. The characteristic features are extensive arterial occlusive disease with the development of corkscrew collateral vessels. It usually affects more than one limb. **Buzzword = Auto-amputation**.

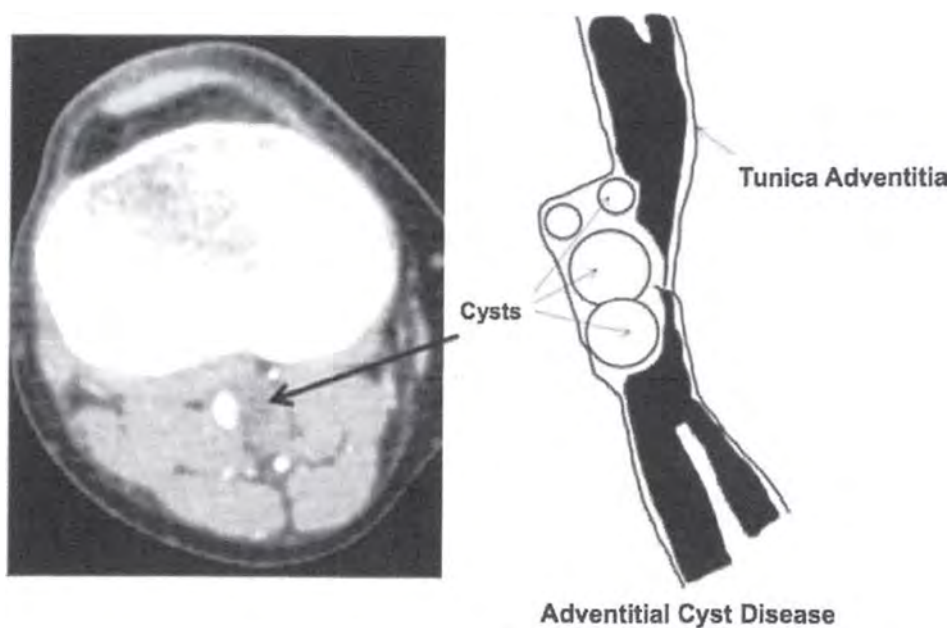
If they are showing you a hand angiogram: It's going to be either Buergers of Hypothenar Hammer Syndrome(HHS). Ask yourself is the ulnar artery involved. If yes go with the HHS. If the ulnar nerve looks ok, but the fingers are out go with Buergers. Be careful, because the fingers can be out with HHS as well (distal emboli). Pseudo-aneurysm off the ulnar artery is a slam dunk for HHS.

Large Vessel	
Takayasu	Young Asian Female - thickened aneurysmal aorta
Giant Cell	Old Person with involvement of the crutches (Subclavian, axillary, brachial).
Cogan Syndrome	Kid with eye and ear symptoms + Aortitis
Medium Vessel	
PAN	More common in man. Renal Micoraneurysm (similar to speed kidney). Associated with Hep B.
Kawasaki	Coronary Artery Aneurysm
Small Vessel (ANCA+)	
Wegeners	Nasal Septum Erosions, Cavitary Lung Lesions
Churg Stauss	Transient peripheral lung consolidations.
Microscopic Polyangitis	Diffuse pulmonary hemorrhage
Small Vessel (ANCA -)	
HSP	Kids. Intussusception. Massive scrotal edema.
Behcets	Pulmonary artery aneurysm
Buergers	Male smoker. Hand angiogram shows finger occlusions.

Misc:

SAM (Segmental Arterial Mediolysis) - Affects the splanchnic arteries in the elderly, and the coronaries in young adults. Not a true vasculitis, with no significant inflammation. It's complicated but essentially the media of the vessel turns to crap, and you get a bunch of aneurysms. The aneurysms are often multiple. The way this is shown is **multiple abdominal splanchnic artery saccular aneurysms** - *this is the disease hallmark.*

Cystic Adventitial Disease - This uncommon disorder classically **affects the popliteal artery, of young men**. Basically you have one or **multiple mucoid filled cysts** developing in the outer media and adventitia. As the cysts grow they compress the artery.



Carotid Doppler

There are a couple of high yield topics regarding carotid Doppler.

Stenosis: They will show you an elevated velocity (normal is 125cm/s). They may also show you ICA/CCA ratio (normal is 2), or the ICA end diastolic velocity (< 40 is normal).

Here are the rules:

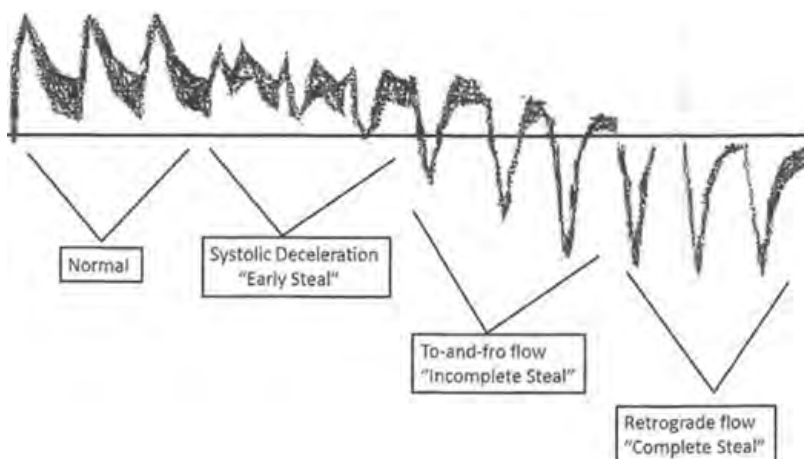
- o 50-69% Stenosis: ICA PSV 125-230cm/s , ICA/CCA PSV ratio: 2.0-4.0 , ICA EDV 40-100
- o >70 % Stenosis: ICA PSV > 230cm/s, ICA/CCA PSV ratio: >4.0 , ICA EDV >100

Proximal Stenosis: OK here is the trick; they will show tardus parvus waveform. **If they show it unilateral, it is stenosis of the innominate. If it's bilateral then it's aortic stenosis.**

Subclavian Steal: This is discussed in greater detail in the cardiac chapter, but this time lets show it on ultrasound. As a refresher, we are talking about stenosis and/or occlusion of the proximal subclavian artery with retrograde flow in the ipsilateral vertebral artery.

How will they show it? They are going to show two things: (1) Retrograde flow in the left vertebral, and (2) a stenosis of the subclavian with a high velocity.

How they can get really sneaky? They can show this thing called “early steal.” Steal is apparently a spectrum, which starts with mid-systolic deceleration with antegrade late-systolic velocities. Some people think the “early steal” waveform looks like a rabbit.



Aortic Regurgitation: - Just like aortic stenosis they are going to show you bilateral CCAs. In this case you are going to get **reversal of diastolic flow**.

Internal Carotid vs External Carotid: - This really lends itself well to multiple choice test questions. The big point to understand is that the brain is always on. You need blood flow to the brain all the time, which means diastolic flow needs to be present all the time, and thus continuous color flow throughout the cardiac cycle. The external carotid feeds face muscles... they only need to be on when you eat and talk.

Internal Carotid	External Carotid
Low Resistance	High Resistance
Low Systolic Velocity	High Systolic Velocity
Diastolic velocity does not return to baseline	Diastolic velocity approaches zero baseline
Continuous color flow is seen throughout the cardiac cycle	Color flow is intermittent during the cardiac cycle

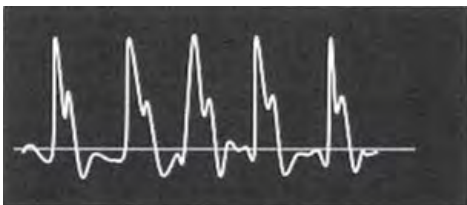
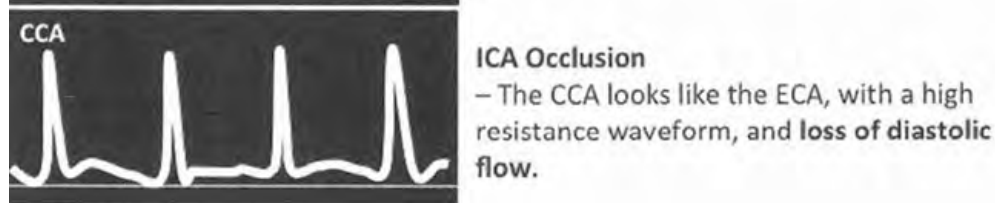
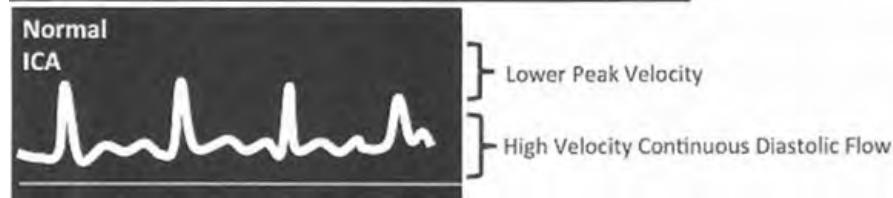
Temporal Tap - This is a point of trivia, that I guess one can perform. It is a technique sonographers use to tell the external carotid from the internal carotid. You tap the temporal artery on the forehead and you see ripples in the spectrum.

As a point of trivia, you can also **look for branches to tell the external carotid vs the internal.*

Brain Death - Apparently in Europe ultrasound can be used for brain death studies. **A loss of diastolic flow suggests cessation of cerebral blood flow.**

Aneurysms: - In case someone asks you, distal formation of an aneurysm (such as one in the skull) cannot be detected by ultrasound, because proximal flow is normal.

Classic Carotid Doppler Cases



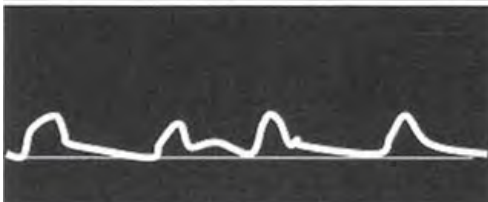
Aortic Regurgitation

- With Classic Reversal of Diastolic Flow
- Most likely shown bilaterally.



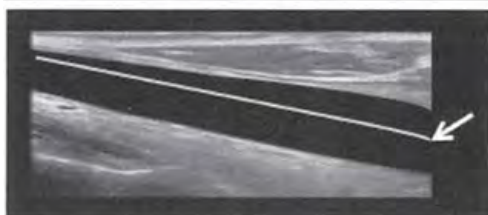
Aortic Regurgitation

- This time showing the "*Pulsus Bisferiens*", or double systolic peak. This is also seen in severe COPD.



Aortic Stenosis

- Characteristic Tardus Parvus Waveform
- This will be shown **bilateral** - to prove it's the valve. Unilateral will be a more central vascular stenosis.



Dissection - with Flap

9 Interventional

Prometheus Lionhart, M.D.



Interventional Radiologists are very good at many things. However, they tend to be terrible at making multiple choice test questions out of the complex dynamic procedures they do on a daily basis. As a result, IR questions tend to fall into one of two categories: (1) what is this very basic thing, number, or anatomy structure ? or (2) if you were me and I was doing this case what would I be thinking next? Obviously, category one tends to make up the majority of the questions and category two has such bad statistics that they would likely get tossed out.

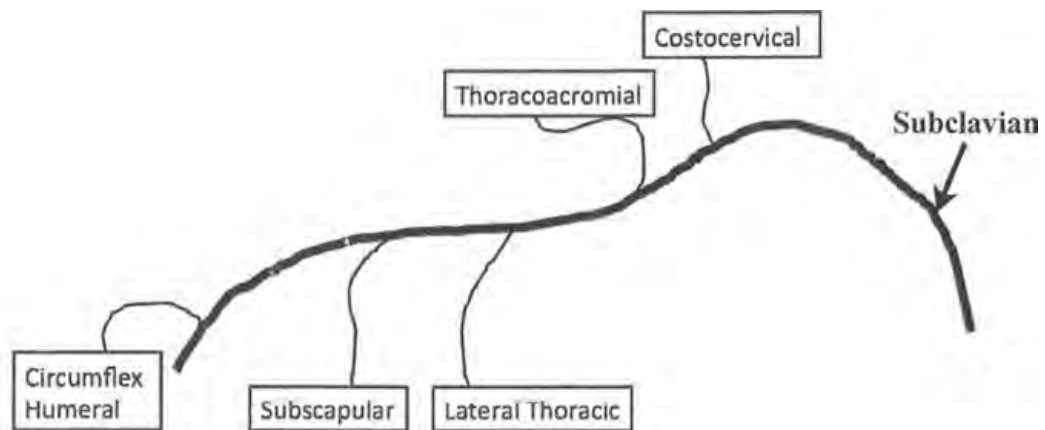
High Yield Topics:

- Anatomy
- French Size, Sheath Size, Inner and Outer Lumen Size,
- Conversion from mm to inches
- Indications / Contraindications to procedures

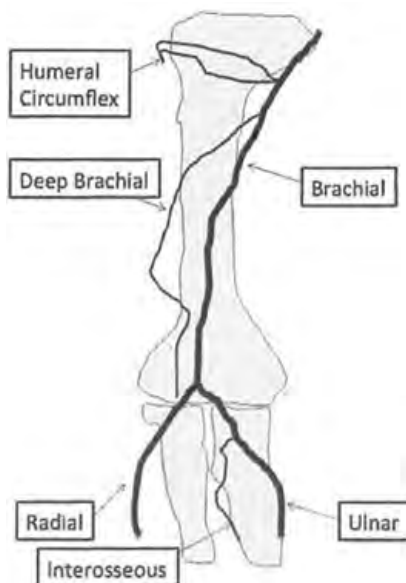
Anatomy Review

Upper Extremity Anatomy:

Remember that the subclavian artery runs posterior to the subclavian vein. The artery has several major branches: the vertebral, the internal thoracic, the thyrocervical trunk, the costocervical trunk, and the dorsal scapular.



Terminology:



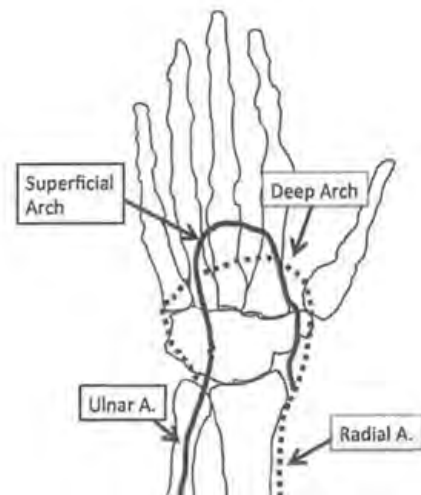
‘Subclavian Artery - From the Brachiocephalic to the First Rib (outer edge)

•**Axillary Artery** - From the First Rib (outer edge) to the Teres Major (outer edge)

‘Brachial Artery - From Teres Major (outer edge) to bifurcation of the radial and ulnar arteries

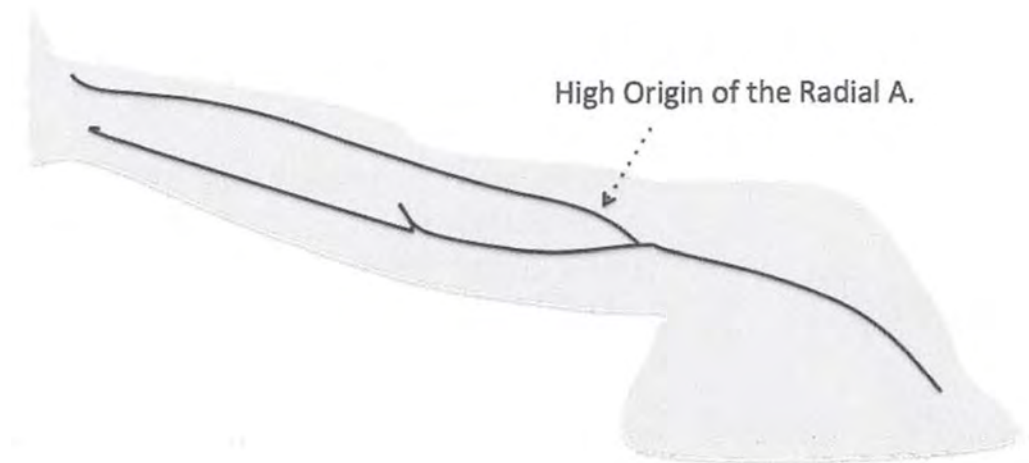
Arm: Around the radial head the brachial artery splits into the radial and ulnar arteries. The recurrent radial and anterior / posterior ulnar recurrents form just beyond their respective origins . Usually, the ulnar artery is larger than the radial and gives off the common interosseous - which then splits off to form anterior and posterior branches.

Hand: The arterial anatomy of the hand is highly variable, with the ulnar artery supplying the superficial arch and the radial artery supplying the deep arch.



Normal Variants:

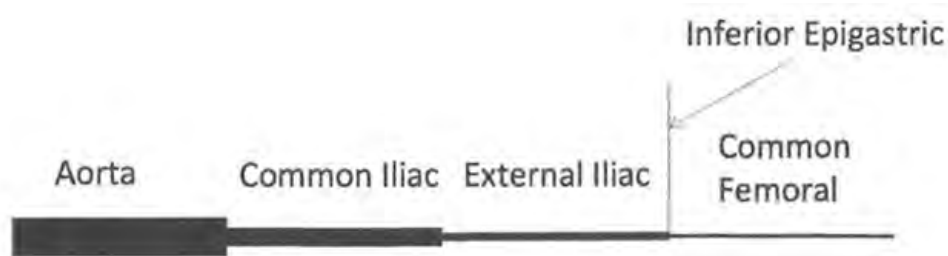
- * Anterior Interosseous Branch (Median Artery) persists and supplies the deep palmar arch of the hand.
- * **“High Origin of the Radial Artery”** - Radial artery comes off either the axillary or brachial artery



Pelvic / Lower Extremity Anatomy:

Pelvic Vascular Anatomy - The pelvic vascular anatomy is super high yield and lends well to multiple choice tests so I want to try and explain it as clearly as possible.

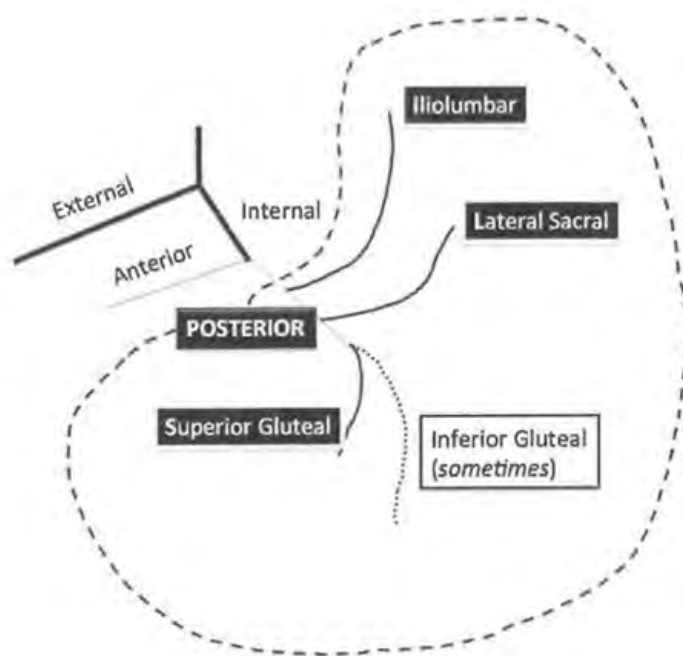
Every medical student knows the aorta bifurcates into the right and left common iliac arteries, which subsequently bifurcate into the external and internal iliac arteries. The nomenclature pearl for **the external iliac is that it becomes the common femoral once it gives off the inferior epigastric** (*at the inguinal ligament*).



The internal iliac is more tricky. It first divides into anterior and posterior divisions.

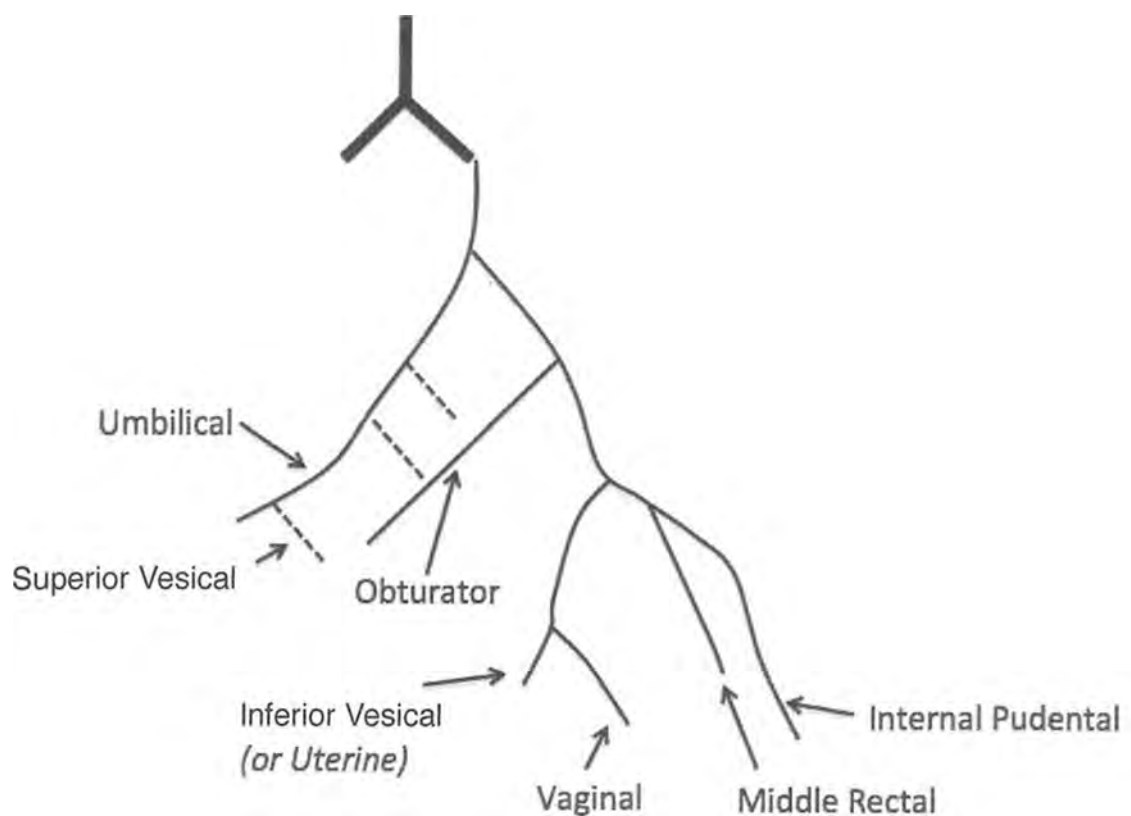
Posterior Division:

The posterior divisions are easier to remember because there are only 3 (sometimes 4) branches. There are two small branches (Iliolumbar and Lateral Sacral), with the terminal Superior Gluteal. The **mnemonic “I-Like - Sex”** is the one I use to remember these. Sometimes the inferior gluteal comes off the posterior division, and this screws up my mnemonic.



Anterior Division:

The anterior division has more branches and is more complicated. The first branch is a slender “umbilical” artery that heads toward the anterior umbilical wall. It eventually becomes obliterated (*obliterated umbilical artery*), before running into the anterior umbilical wall (*medial umbilical ligament*). The umbilical artery gives off numerous branches that supply the fundus of the bladder (superior vesical arteries). The second branch is the obturator artery, which runs through the obturator canal to supply muscles in the medial thigh. In a man, the third branch is the inferior vesical which supplies the base of the bladder and prostate. The inferior vesical is absent in women, and instead you have the uterine artery. The uterine artery gives birth to the vaginal artery. The anterior division terminates by giving off two branches, the middle rectal artery and the internal pudendal artery (which supply external genitals).



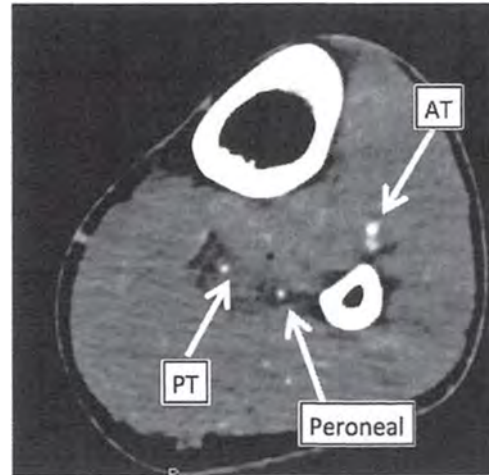
Anterior Division

Lower Extremity Vascular Anatomy

As mentioned above, once the inferior epigastric comes off (level of the inguinal ligament) you are dealing with the common femoral artery (CFA).

The CFA divides into the deep femoral (profunda) and superficial femoral. The deep femoral courses lateral and posterior. The superficial femoral passes anterior and medial into the flexor muscle compartment (Adductor / Hunter's Canal). At the point the vessel emerges from the canal it is then

the popliteal artery. At the level of the distal border of the popliteus muscle the popliteal artery divides into the anterior tibial (the first branch) and the tibial peritoneal trunk. In case someone would ask you, the anterior tibial courses anterior and lateral, then it transverse the interosseous membrane, running down the front of the anterior tibia and terminating as the dorsalis pedis. The tibial peroneal trunk bifurcates into the posterior tibial and fibular (peroneal) arteries. A common quiz is "what is the most medial artery in the leg?", with the answer being the posterior tibial (felt at the medial malleolus). Notice how lateral the AT is. You can imagine it running across the interosseous membrane, just like it's suppose to.

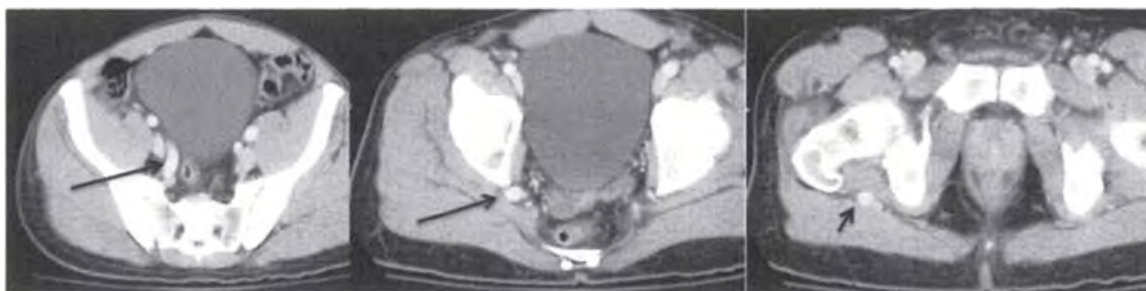


Variant Anatomy:

Persistent Sciatic Artery - An aberrant vessel that comes off the internal iliac, passes through the sciatic foramen and joins the popliteal artery just above the knee to be the dominant source of flow to the lower leg.

Trivia About Persistent Sciatics:

- (1) The SFA is often absent or hypoplastic
- (2) It's prone to intimal injury / aneurysm formation



Persistent Sciatic Artery

GI Section

Biliary Duct Anatomy Trivia: The right posterior segment branch draining the left hepatic duct, is the most common ductal variant. The second most common is trifurcation of the intrahepatic radicles.

G Tubes: A “G- Tube” is a gastric tube, placed directly into the stomach. The basic idea is that you put an NG tube down and pump air into the stomach until it smushes flat against the anterior abdominal wall. Then you spear it and secure it with 4 “T-Tacks” to retract the stomach to the abdominal wall in the high gastric body. Then spear it again, wire in and dilate up to the size you want. Typically, the T-Tacks are removed in 3-6 weeks. Other things that you can do is give a cup of barium the night before to outline the colon. If the patient has ascites it’s good form to drain that first. You can do it under ultrasound or CT. Honestly, it’s probably best to do it with a scope, but since we are Radiologists my official statement is that only a Radiologist can do this procedure well.

Abscess Drainage: There are two methods, you can use a trocar or you can use the seldinger technique (wire guided).

- * t rocar: You nail it with a spinal needle first. Then adjacent to the needle (in tandem) you place a catheter.
- * Seldinger: One stick with a needle, then wire in, dilate up and place a catheter.

Liver Abscess - Lots of etiologies for these, but don’t forget to think about the appendix or diverticulitis. The draining of these things is somewhat controversial with some authors feeling the risk of peritoneal spread out weighs the benefits and reserving the drainage for patient’s with a poor prognosis. Other authors say that everyone and their brother should get one, and consider it first line treatment. A pearl to draining these things is to not cross the pleura (you’ll give the dude an empyema). If there is a biliary fistula, prolonged drainage will usually fix it (biliary drainage or surgery is rarely needed).

Biliary Drainage - The ductal anatomy mimics the segmental anatomy. The simple version is at the hilum. There are two main hepatic ducts (right and left) which join to make the common hepatic duct. The right hepatic duct is made of the horizontal right posterior (segment 6 & 7) and vertical right anterior (segment 5 & 8). The left duct has a horizontal course and drains segment 2 and 4.

The role of PTC (Percutaneous Transhepatic Cholangiogram) and PTBD (Percutaneous Transhepatic Biliary Drainage) is centered around situations when ERCP and endoscopy have failed or are not possible (Roux-en-Y).

Antibiotics are important to give first (ascending cholangitis is bad). There are two approaches; right lateral mid axillary for the right system, or subxyphoid for the left system. Realistically diagnostic cholangiogram and PTBD is usually done from the right. The left is more technically challenging (although better tolerated by the patient because the tube isn't in between ribs) and usually there is a hilar stricture that won't allow the left and right system to communicate. Basic idea is to randomly jam a chiba needle in and inject slowly under fluoro as you pull back. Once you get into a duct the system will opacify. You then can pick your target (posterior is best for best drainage). You stick again, wire in, and place the catheter into the duodenum. A non-dilated system can be very difficult and there is an old school trick where you stick the gallbladder (on purpose) and retrograde fill the system. The problem with that is you have to keep a drain in the gallbladder as well.

Random Trivia:

- * *During PTC - if you encounter (or expect stones), dilute contrast to 200-250mg/ml to avoid obscuring filling defects.*
- * *Forceful Injection = ICU Visit for Cholangitis*
- * *You can make 15-20 passes trying to get into a bile duct, after that most texts say you should quit*

Cholecystostomy: This is done when you have a super sick patient you can't take to the OR, but has a toxic gallbladder. In cases of acalculous cholecystitis (with no other source of sepsis), 60% of the time cholecystostomy is very helpful. It's a "temporizing measure." You have to give pre-procedure antibiotics. There are two approaches:

- * **Transperitoneal** - This is preferred by many because it's a direct approach, and avoids hitting the liver. The major draw back is the wire / catheter often buckles and you lose access (and spill bile everywhere).
- * **Transhepatic** - The major plus here is that when you cross the liver it stabilizes the wire and **minimizes the chance of a bile leak.**

Important Trivia:

- * Even if the procedure instantly resolves all symptoms, you need to leave the tube in for 2-6 weeks (until the tract matures), otherwise you are going to get a bile leak.

Managing Bile Leak - Bile leak is bad, it can cause massive biliary ascites and chemical peritonitis. You can place a tube within the bile ducts to divert bile from the location of the leak (this usually works).

TIPS (Transjugular Intrahepatic Portosystemic Shunt) -

What is this portal hypertension? The portal vein gives you 70-80% of your blood flow to the liver. The pressure difference between the portal vein and IVC (“PSG”, portal systemic gradient) is normally 3-6 mm Hg. Portal HTN is defined as pressure in the portal vein > 10mm Hg or PSG > 6mm Hg. The most common cause is EtOH (in North America).

What does portal hypertension look like? On ultrasound we are talking about an enlarged portal vein (>1.3-1.5cm), and enlarged splenic vein (> 1.2cm), big spleen, ascites, portosystemic collaterals (umbilical vein patency), and reversed flow in the portal vein.

Who gets a TIPS ? Accepted indications include variceal hemorrhage that is refractory to endoscopic treatment, and refractory ascites. The second part of this question is answered by a MELD (bilirubin, INR, creatinine) score. MELD scores greater than 18 are at higher risk of early death after an elective TIPS.

Preprocedural steps for TIPS? You need two things. (1) An ECHO to evaluate for heart failure (right or left). (2) Cross sectional imaging to confirm patency of the portal vein.

How is a TIPS done? The real answer is do an IR fellowship.

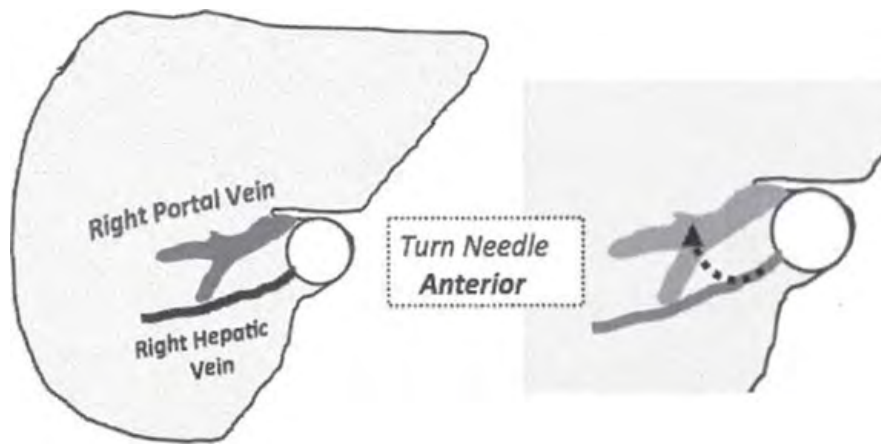
First thing you do is measure the right heart pressure. If it is elevated (10-12 mmHg) you stop. A normal right heart pressure is around 5.

If it is normal, you proceed with the procedure. Access the jugular vein on the right, go down the IVC to the hepatic veins, opacify the veins, do a wedge pressure (don’t blow the capsule off), use CO₂ to opacify the portal system.

Then stick “Crotch to Crotch” from the hepatic veins to the portal vein (usually right to right). Then put a stent in and balloon it up. Lastly check pressures and make you sure you didn’t over do it (usually want a gradient around 9-12).



H hit It diteitiott do you turn the catheter when you are moving from the right hepatic vein, to the ti Jit portal vein? You want to turn anterior.



The main acute post procedural complications of TIPS include: cardiac decompensation (elevated right heart filling pressures), accelerated liver failure, and worsening hepatic encephalopathy.

Evaluation of a “Normal TIPS ”

Because the stent decompresses the portal system, you w'ant to see flow directed into the stent. Flow should reverse in the right and left portal vein and flow directly into the stent. Flow in the stent is typically 90-190cm.

Stenosis / Malfunction:

- * Elevated maximum velocities ($> 200\text{cm/s}$) across a narrowed segment.
- * Low portal vein velocity ($< 30\text{cm/s}$ is abnormal).
- * A temporal increase (or decrease) in shunt velocity by more than 50 cm/s is also considered direct evidence.
- * “Flow Conversion” with a change of flow in a portal vein branch towards the stent to away from the stent.
- * An indirect sign of malfunction is new or increased ascites.

Addressing Hepatic Encephalopathy - Dropping the gradient too low increases the risk of HE. If the TIPS is too open you may need to tighten it down with another stent.

What are the absolute contraindications for TIPS? Severe heart failure (right or left), biliary sepsis, isolated gastric varices with splenic vein occlusion. Relative contraindications include cavernous transformation of the portal vein, and severe hepatic encephalopathy.

What is an alternative to TIPS for treatment of refractory ascites? There is a rarely indicated thing called a “peritoneovenous shunt.” This stupid thing has a high rate of infection and thrombosis, and can even lead to DIC. It’s designed to allow drainage of the ascites through a tunneled line all the way up to the systemic circulation (jugular).

BRTO (Balloon-Occluded Retrograde Transverse Obliteration).

TIPS and BRTO are brother and sister procedures in what they treat and what they do:

TIPS	BRTO
Treat Esophageal Varices	Treat Gastric Varices
Place a shunt to divert blood around liver	Embolize collaterals drives blood into liver
Complication is worsening hepatic encephalopathy	Complication is worsening esophageal varices and worsening ascites
Improves esophageal varices and ascites	Improves hepatic encephalopathy

The idea is that you access the portosystemic gastroduodenal shunt from the left renal vein via a transjugular or transfemoral approach. A balloon is used to occlude the outlet of either the gastroduodenal or gastro-caval shunt. Following balloon occlusion, a venogram is performed. A sclerosing agent is used to take the vessels out. After 30-50 minutes you aspirate the remaining sclerosing agent and let down the balloons.

As a point of trivia, the most common side effect of BRTO is gross hematuria.

Liver biopsy - You can do targeted approaches (for a specific lesion) or you can do non-targeted approaches (sampling). General pearls include; trying to cross the capsule only once, biopsy of subcapsular masses through an area of uninvolved liver and avoiding the diaphragm.

Uncorrectable coagulopathy, thrombocytopenia ($< 50,000$), infections in the right upper quadrant - are contraindications for a conventional biopsy. In cases of massive ascites or severe coagulopathy a transjugular approach can be performed.

Complications are rare, and bleeding is the one you worry about the most. Shoulder pain and a “patent track” after 5 minutes can make you more vigilant for a bleed. Always look behind the liver (Morrison’s pouch) to see if blood is accumulating. Bleeding after liver biopsy occurs more from biopsy of malignant lesions (compared to diffuse disease). Note that biopsy of carcinoid mets is controversial and death by carcinoid crisis has occurred after biopsy.

GI Bleeding - You can split GI bleeds into two categories upper (proximal to ligament of Treitz) and lower.

Vasopressin: First line treatment is usually vasopressin injection. Contraindications to vasopressin include: large artery bleeding (i.e. splenic pseudoaneurysm), bleeding at sites with dual blood supply (classic example is pyloroduodenal bleed), severe coronary artery disease, severe hypertension, dysrhythmias, and after an embolotherapy treatment (risk of bowel infarct).

High Yield Trivia is that **nuclear scintigraphy (RBC bleeding scan) is more sensitive than angiography.**

- * GI Bleed Scan = 0.1 mL/min
- * Angiography = 1.0mL/min

Embolization: This is used for patients who fail (or have contraindication) to vasopressin. The most common tools are coils or gelfoam. Contraindications / reasons for increased risk include; prior surgery (stomach or bowel), prior radiation, or if the collateral circulation is not adequate. Another piece of trivia is the need to do angiography post embolization to look for collateral flow (if there is a dual supply). The classic example is: after performing an embolization of the GDA (for duodenal ulcer), you need to do a run of the SMA to look at the inferior pancreaticoduodenal. You might have to take that one out too, but obviously that would increase the risk of bowel infarct.

Upper GI: Some testable trivia is that 85% of upper GI bleeds are from the left gastric, and often if a source cannot be identified the left gastric is taken down prophylactically. If the source of bleeding is from a duodenal ulcer, embolization of the GDA is often performed. About 10% of the time, an upper GI bleed can have bright red blood per rectum.

Lower GI: The most common cause is diverticulosis (usually left sided). Embolization of angiodysplasia rarely stops a re-bleed and these often need surgery.

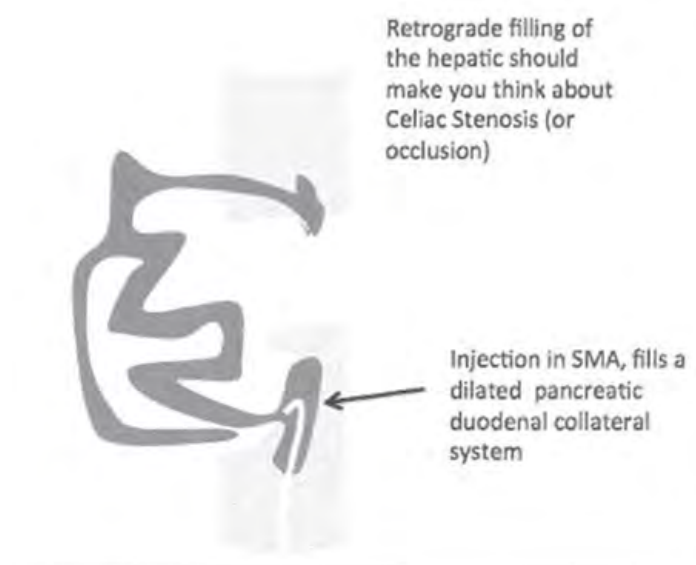
“Pseudo-Vein ” Sign - This is a sign of active GI bleeding, with the appearance of a vein created by contrast pooling in a gastric rugae or mucosal intestinal fold. If you aren’t sure if it’s an actual vein, the “pseudo-vein” will persist beyond the venous phase of injection.

Dieulafoy’s Lesion - This is a monster artery in the submucosa of the stomach which pulsates until it causes a teeny tiny tear (not a primary ulcer). These tears can bleed like stink. It’s typically found in the lesser curvature. It’s not exactly an AVM, more like angiodysplasia. Sometimes you can treat it with clips via endoscopy. Sometimes it needs endovascular embolization.

When I say pancreatic arcade bleeding aneurysm, you say celiac artery stenosis.

There is a known association with celiac artery compression (median arcuate ligament) and the dilation of pancreatic duodenal arcades with pseudoaneurysm formation.

Gamesmanship: It is classically shown with an angiographic run through the SMA, showing a dilated collateral system and retrograde filling of the hepatic artery.



HCC Treatment: Transplant remains the only way to “cure” an HCC, but IR offers several palliative ways to address it including TACE, and percutaneous ablations (ethanol, microwave, RFA).

Transarterial Chemoembolization (TACE) - This is the mainstay of palliative therapy for HCC (transplant is the only “cure”). Using an iodized oil as a transport agent for anticancer drugs. This causes an acute ischemia to the HCC lesion, and coagulative necrosis (since HCC’s drink arterial blood). On follow up CT, you need to have pre and post contrast imaging including washout. The iodized oil is going to be dense on the pre-contrast. The necrotic tissue should not enhance. If there is enhancement and/or washout out in or around the tumor, then you have viable tumor that needs additional treatment. Beam hardening from the iodized oil can cause a problem.

RFA: Tumor is destroyed by heating the tissue to 60 degrees C. Any focal or nodular peripheral enhancement in the ablation lesion should be considered residual / recurrent disease. Sometimes, on the immediate post treatment study you can have some reactive peripheral hyperemia - but this should decrease on residual studies. Important trivia is that RF ablation is indicated in patients with HCC and colorectal mets (who can’t get surgery).

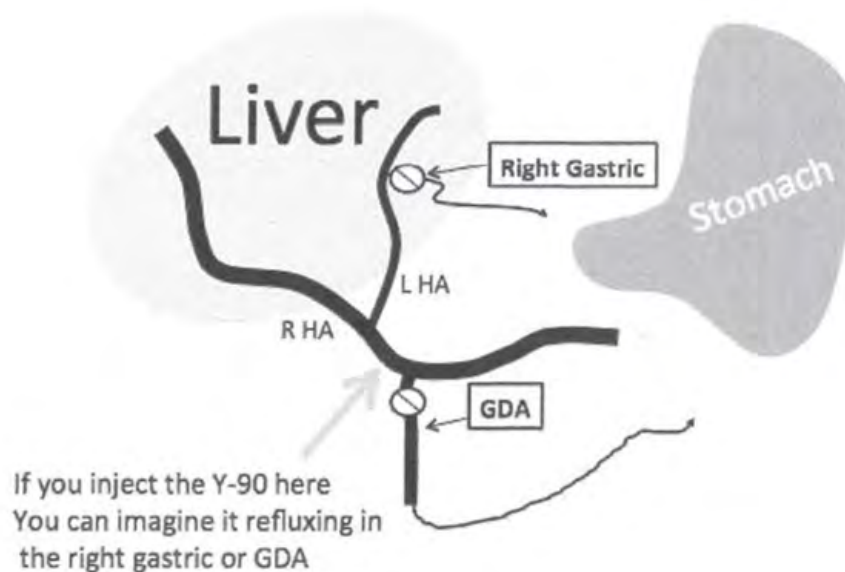
TACE + RFA: As a point of trivia, it has been shown that TACE + RFA for HCC lesions larger than 3cm, will improve survival (more so than either treatment alone). This is still not curative.

Trivia:

- * *TACE will prolong survival better than systemic chemo*
- * *Portal Vein Thrombosis is considered a contraindication (sometimes) because of the risk of infarcting the liver.*
- * *"Zone of Ablation " is the preferred nomenclature for the post-ablation region on imaging.*

Yttrium-90 Radioembolization - An alternative to TACE is using radioactive embolic materials (Y-90). The primary testable trivia regarding Y-90 therapy is understanding the pre-therapy work up. There are basically two things to know:

- (1) *Lung Shunt Fraction* - You give Tc-99 MAA to the hepatic artery to determine how much pulmonary shunting occurs. A shunt fraction that would give 30 Gy in a single treatment is too much (Y-90 is contraindicated).
- (2) *The take off of the right gastric*. The fear is that you get non-targeted poisoning of the stomach, leading to a non-healing gastric ulcer. To help prevent reflux of the Y-90 (poison) into places you don't want (basically anywhere that's not liver) prophylactic embolization of the right gastric and the GDA is performed. The right gastric origin is highly variable, and can come off the proper hepatic or the left hepatic.



Hepatic / Splenic Trauma - Embolization is the primary method for dealing with significant trauma to the hepatic or splenic arteries. In the liver, you can embolize directly at the site of injury (because of the dual blood supply). In the spleen there are several options. In the situation of a splenic pseudo aneurysm from intrasplenic vessels, you can do selective embolization at the point of injury with the idea that this preserves splenic tissue (risk is focal infarction). In the situation of active bleeding in or beyond the spleen, embolize the proximal splenic artery, and reduce the pressure to the spleen (slower blood will clot), with the benefit of preserved collateral supply and less embolization risk. The patient usually does not require vaccination post embolization, as a lot of functional tissue should remain .

Thoracic Section

Pleural Drainage - Most everyone has done a few thoracenteses as a resident. I just want to touch on a few testable points. (1) Remember that you go “above the rib” to avoid the neurovascular bundle. (2) If you pull off too much fluid too fast you can possibly get pulmonary edema from re-expansion (this is uncommon). (3) If it’s malignant you might end up with a trapped lung (lung won’t expand fully). A “vacu-thorax” does not mean anything, and does not need immediate treatment even if it’s big. If you really need to fix it, you’ll need a surgical pleurectomy / decortication. Pleurodesis (which can be done to patients with recurrent pleural effusions), does NOT help in the setting of trapped lung. (4) Pneumothorax is rare but is probably the most common complication (obviously it’s more common when done blind).

Lung Abscess: Just remember that you can drain an empyema (pus in the pleural space), but you should NOT drain a lung abscess because you can create a bronchopleural fistula (some people still do it).

Lung Biopsy - The most common complication is pneumothorax, which occurs about 25% of the time (most either resolve spontaneously or can be aspirated), with about 5% needing a chest tube. The second most common complication (usually self-limiting) is hemothysis. The testable pearls include: (1) the lower lung zones are more affected by respiratory motion, (2) The lingula is the most affected by cardiac motion, (3) avoid vessels greater than 5mm, (4) try and avoid crossing a fissure (they almost always get a pneumothorax), (5) Areas lateral to and just distal to the tip of a biopsy gun will be affected by “shock wave injury”, so realize vessels can still bleed from that.

Reducing the Risk of Pneumothorax - Post Biopsy

Avoid interlobar fissures

Put the patient puncture side DOWN after the procedure

No talking or deep breathing after the procedure (at least 2 hours)

If the patient is a cougher, consider postponing the procedure - or giving empiric anti-tussive meds

When to Place a Chest Tube? - Post Biopsy

Pneumothorax is symptomatic

Pneumothorax continues to enlarge on serial radiographs

Lung RFA - Radiofrequency ablation of lung tumors can be performed on lesions between 1.5cm and 5.2cm in diameter. The most common complication is pneumothorax (more rare things like pneumonia, pseudoaneurysm, bronchopleural fistula, and nerve injury have been reported). The effectiveness of RFA is similar to external beam radiation with regard to primary lung cancer. The major advantage of lung RFA is that it has a limited effect on pulmonary function, and can be performed without concern to prior therapy.

Imaging (CT and PET) should be performed as a follow up of therapy. Things that make you think residual /recurrent disease: nodular peripheral enhancement measuring more than 10 mm, central enhancement (any is bad), growth of the RFA zone after 3 months (after 6 months is considered definite), increased metabolic activity after 2 months, residual activity centrally (at the burned tumor).

Occlusion of Central Veins (SVC Syndrome) - There are a variety of ways to address occlusion of the SVC. The goal is to return in line flow from at least one jugular vein, down through the SVC. Most commonly thrombolysis is the initial step, although this is rarely definitive. The offending agent (often a catheter) should be removed if possible. If the process is non-malignant, often angioplasty alone is enough to get the job done (post lysis). In malignant causes, you should do lysis, then angioplasty, then stent. Non-malignant causes may still need a stent if the angioplasty doesn't remove the gradient (if the collateral veins are still present). Self expanding stents should NOT be used, as they tend to migrate. The last pearl on this one is not to forget that the pericardium extends to the bottom part of the SVC and that if you tear that you are going to end up with hemopericardium and possible tamponade.

Thoracic Angio: The basic idea is that the primary indication for pulmonary arteriography is diagnosis and treatment of PE. The “Grollman” catheter, which is a preshaped 7F, is the classic tool. You get it in the right ventricle (usually from the femoral) and then turn it 180 degrees so the pigtail is pointing up, then advance it into the outflow tract. Cardiac dysrhythmias may occur and you should re-position the catheter / wire if you get a short run of v-tach. Some people say that a **known LBBB is high risk**, and these patients should get prophylactic pacing (because the wire can give you a RBBB, and RBBB + LBBB = asystole). An important thing to know is that patients with chronic PE often have pulmonary hypertension. Severe pulmonary hypertension needs to be evaluated before you inject a bunch of contrast. **Pressures should always be measured before injecting contrast** because you may want to reduce your contrast burden. Oh, one last thing about angio... never ever let someone talk you into injecting contrast through a swan-ganz catheter. It’s a TERRIBLE idea and the stupid catheter will blow apart at the hub.

Contraindications to Pulmonary Angiography

There are two major contraindications:

- (1) **Pulmonary HTN** with elevated right heart pressures (greater than 70 systolic and 20 end diastolic).

If you need to proceed anyway - they get low osmolar contrast agents injected in the right or left PA (NOT the main PA).

- (2) **Left Bundle Branch Block** - The catheter in the right heart can cause a right block, leading to a total block.

If you need to proceed anyway - they get prophylactic pacing.

Pulmonary AVM - They can occur sporadically. For the purpose of multiple choice when you see them think about HHT (Hereditary Hemorrhagic Telangiectasia / Osier Weber Rendu). Pulmonary AVMs are most commonly found in the lower lobes (more blood flow) and can be a source of right to left shunt (**worry about stroke and brain abscess**). The rule of **treating once the afferent vessel is 3mm** is based on some tiny little abstract and not powered at all. Having said that, it’s quoted all the time and a frequent source of trivia that is easily tested.

Pulmonary Embolism - Patients with PE should be treated with medical therapy (anticoagulation with Coumadin, Heparin, or various newer agents), allowing the emboli to spontaneously undergo lysis. In patients who can't get anticoagulation (for whatever reason), IVC filter should be placed. The use of transcatheter therapy is typically reserved for unstable patients with massive PE. In those situations, catheter directed thrombolysis, thromboaspiration, mechanical clot fragmentation, and stent placement have all been used to address large clots.

Hemoptysis - Massive hemoptysis (> 300cc) can equal death. Bronchial artery embolization is first line treatment (bronchial artery is the culprit 90% of the time). Unique to the lung, active extravasation is NOT typically seen with the active bleed. Instead you see tortuous, enlarged bronchial arteries. The main thing to worry about is infarcting the cord. Thus a large spinal artery or radiomedullary branch from the target artery is a contraindication (according to some people). Also, "hairpin-shaped" anterior medullary arteries are a contraindication (arise from intercostal bronchial trunk in 10% of the cases). Particles (> 325 micrometers) are used (coils should be avoided).

Hemoptysis Pearls:

- * *The bronchial arteries are usually the source*
- * *Remember bronchial arteries don't "blush" the way active bleeders in other parts of the body do. You are looking for an enlarged tortuous vessel.*
- * *Pulmonary Artery is usually NOT the source (unless it's a traumatic pseudoaneurysm from a Swan, Pulmonary AVM, or Rasmussen Aneurysm from TB).*
- * *The most feared complication is transverse myelitis from an accidental plugging of an anterior spinal artery feeder. Avoid the "hairpin shaped" vessels.*
- * *Any cause of chronic lung inflammation (sarcoid, CF, TB) will cause occlusion of pulmonary arterioles and lead to hypertrophy of the bronchial circulation (which can bleed). **
- * *Never treat a bleeding bronchial artery with coils. It will re-bleed and you will be jailed out. The exception to this rule is aneurysms and AVMs.*

Reproductive / Endocrine Section

Uterine Artery Embolization (UAE): This has been around along time and can be used for bleeding or the bulk symptoms of fibroids. Occlusion of small feeding arteries cause fibroid infarction (and hopefully shrinkage). Embolic material is typically PVA or embospheres for fibroids and gel foam or glue for post partum hemorrhage / vaginal bleeding.

Trivia:

- * Gonadotropin-releasing medications (often prescribed for fibroids) should be stopped for 3 months prior to the case (they cause constriction of the uterine artery).
- * Remember the uterine artery is off the anterior division of the internal iliac
- * Regardless of the fibroid location, bilateral embolization is necessary to prevent recruitment of new vessels
- * In most cases branches of the ovarian artery feed the fibroids via collaterals with the main uterine artery
- * Submucosal lesions and smaller tumors tend to have the best response
- * “Cellular” Fibroids , which are densely packed smooth muscle (without much connective tissue) and high T2 signal tend to respond well to embolization
- * Treatment of adenomyosis with UAE is done exactly the same way, and is an effective treatment for symptomatic relief (although symptoms recur in about 50% of the cases around 2 years post treatment).
- * The EMMY trial showed that hospital stays with UAE are shorter than hysterectomy
- * The incidence of premature menopause is around 5%
- * DVT / PE is a known risk of the procedure (once pelvic vein compression from large fibroid releases - sometimes the big PE flies up). The risk is about 5%.

Contraindications: Pregnancy, Active Pelvic Infection, Prior Pelvic Radiation, Connective Tissue Disease, Prior Surgery with Adhesions (relative)

Post Embolization Syndrome: Pain, nausea, vomiting, and low grade fever - is basically an expected finding.

Hysterosalpingogram (HSG): I'm 100% certain no one went into radiology to do these things. You do it like a GYN exam. Prep the personal area with betadine, drape the patient, put the speculum in and find the cervix. There are various methods and tools for cannulating and maintaining cannulation of the cervix (vacuum cups, tenaculums, balloons). Insertion of any of these devices is made easier with a catheter and wire. Once the cervix and endometrial cavity have been accessed the contrast is inserted and pictures are obtained.

Contraindications: Pregnancy, Active Pelvic Infection, Recent Uterine or Tubal Pregnancy.

Trivia:

- The ideal time for the procedure is the proliferative phase (day 6-12), as this is the time the endometrium is thinnest (improves visualization, minimizes pregnancy risk).
- It's not uncommon for a previously closed tube to be open on repeat exam (sedative, narcotics, tubal spasm - can make a false positive).
- Air bubbles can cause a false positive filling defect.
- **Intravasation** - The backflow of injected contrast into the venous or lymphatic system, used to be an issue during the Jurassic period (when oil based contrast could cause a fat embolus). Now it means nothing other than you may be injecting too hard, or the intrauterine pressure is increased because of obstruction.
- **The reported risk of peritonitis is 1%.**

Pelvic congestion syndrome - Women have mystery pelvic pain. This is a real (maybe) cause of it. They blame dilated ovarian and periuterine veins in this case, and give it a name ending in the word "syndrome" to make it sound legit. The symptoms of this "syndrome" include pelvic pain, dyspareunia, menstrual abnormalities, vulvar varices, and lower extremity varicose veins. The symptoms are most severe at the end of the day, and with standing.

Diagnosis ? Clinical symptoms + a gonadal vein diameter of 10mm (normal is 5mm).

Treatment ? GnRH agonists sometimes help these patients, since estrogen is a vasodilator. But the best results for treatment of this "syndrome" are sclerosing the parauterine venous plexus, and coils/plugs in the ovarian and internal iliac veins (performed by your local Interventional Radiologist). This is often staged, starting with ovarian veins plugged first, and then (if unsuccessful) iliac veins are plugged second.

Complications ? Complications are rare but the one you worry about is thrombosis of the parent vein (iliac or renal), and possible thrombus migration (pulmonary embolism).

Will it get better on its own ? The symptoms will classically improve after menopause.

Varicocele - They are usually left sided (90%), or bilateral (10%). Isolated right sided varicoceles should prompt an evaluation for cancer (next step = CT Abd).

When do you treat them? There are three indications: (1) infertility, (2) testicular atrophy in a kid, (3) pain.

Anatomy Trivia (regarding varicoceles): Remember that multiple venous collaterals “pampiniform plexus” or “spermatic venous plexus” drain the testicles. Those things come together around the level of the femoral head, forming the internal spermatic vein. The left internal spermatic vein drains into the left renal vein, and the right internal spermatic vein drains into the IVC. Common variants include: multiple veins on the right terminating into the IVC or renal vein, or one right sided vein draining into the renal vein (instead of the IVC).

Why Varicoceles Happen: The “primary factor” is right angle entry of the left spermatic vein into the high pressure left renal vein. Nut-cracker syndrome on the left is another cause (probably more likely asked).

Basic Idea: You get into the renal vein and look for reflux into the gonadal vein (internal spermatic) which is abnormal but confirms the problem. You then get deep into the vein, and embolize close to the varicocele (often with foam), then drop coils on the way back, and often an Amplatzer or other occlusion device at the origin.

Pelvic Abscess Drainage - Drainage of tubo-ovarian abscess, diverticular abscess, or peri-appendiceal abscess can be done a bunch of ways (transabdominal, transgluteal, transvaginal, transrectal). Some people like the transvaginal approach with a trocar because it's one stick, and no dilation. Transrectal approach may be better for very posterior abscesses. Transgluteal approach can also be done for very posterior targets. **The general rule for transgluteal is to avoid the sciatic nerves and gluteal arteries by access through the sacrospinous ligament medially (close to the sacrum, inferior to the piriformis).** A catheter may need to be placed if the collection is too viscous or multi-septated.

When to pull an abscess catheter. As a general rule - when the patient is better (no fever, WBC normal), and output is < 20cc over 24 hours.

When to place an abscess catheter: Just make sure the collection is organized and not leaking all over the place. Some people will say not in the setting of frank peritonitis.

Endocrine

Pancreatic biopsy - Used for differential diagnosis of cystic lesions, atypical cancer, etc. CT or ultrasound can be used. Results are better with larger needles and when the lesion is located in the body or tail. Pancreatitis can occur post biopsy, and is actually more common with biopsy of a normal gland. Tracking cancer through a needle tract is controversial (sorta like a renal biopsy), some people say you shouldn't do it, if resection would be curative.

Pancreas drainage- Remember that necrotizing pancreatitis is bad, but infected necrotizing pancreatitis is a death sentence. So, be careful draining something that is NOT infected already (otherwise you might make it infected). If you aren't sure if it's infected, consider aspirating some for culture (but not placing a tube).

Indications: General indications include infected collections or collections causing mass effects (bowel or biliary obstruction).

Progression to surgery: If you can get 75% reduction in 10 days the drain is good enough. If not, the surgeons can use the tract for a video assisted retroperitoneal debridement (which still avoids open debridement).

Pancreatic Cutaneous Fistula-. Other than pancreatic pseudocysts most pancreatic collections are either brown or grayish. When the fluid is clear, you should think about pancreatic fluid. If this lasts more than 30 days then you have yourself a "persistent pancreatic fistula." Nice job idiot... you could have just left it alone. That will teach you to let those medicine docs pressure you into doing stuff that's not indicated. It may be possible to treat that with octreotide (synthetic somatostatin) to inhibit pancreatic fluid.

Thyroid biopsy - Thyroid nodules are super super common. Biopsy of benign thyroid nodules helps pay the salary of many Radiologists all over the world, and therefore the absurd number needed to biopsy to actually find a cancer is not frequently addressed.

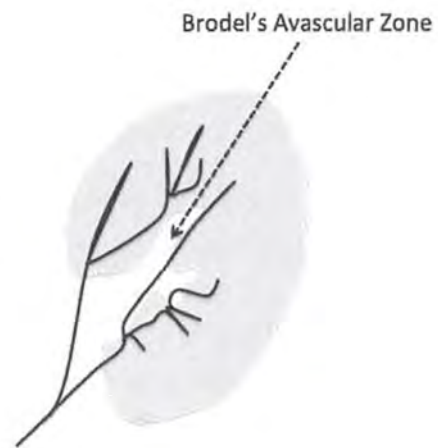
More likely cancer: Things that make a nodules more likely to be cancer are: (a) solid more than cystic, (b) hypervascular, (c) blurred margin, and (d) micro calcifications - this being the most important.

What if the path is not diagnostic? Ultrasound guided FNA is performed, and is most comfortable from the head of the bed. The biopsy usually works, but if you get non-diagnostic results the recommendation is to **not repeat for at least 3 months** as the reparative cellular atypia can complicate results.

Urinary

Percutaneous Nephrostomy (PCN) - This is done for relief of obstruction, diversion for healing (fistula etc.), or to assist with lithotomy. Sepsis would be an indication for emergent PCN. There are only a few true contraindications (bleeding issues, renal cancer). Ethical issues are sometimes listed as a contraindication. An example might be the terminal inoperable patient with irreversible obstruction. The PCN tube can't be removed once it's placed without causing a urine leak. So you are committing this patient to check and changes for the rest of his/her life. Wait? Does he have insurance? Cha-ching \$!

Technique - The lower pole of a posteriorly oriented calyx is ideal. The reason you use a posterior lateral (20-30 degrees) approach is to attack along **Brodeur's Avascular Zone** (area between the arterial bifurcation). Choosing a lower target minimizes the chance of pneumothorax. Additional benefit of the posterior calyces approach is that the guide wire takes a less angled approach (compared to an anterior calyces approach). Direct stick into the collecting system without passing through renal parenchyma is not a good move (high risk of urine leak). The general idea is: ultrasound and stick something, then opacify the system with contrast, use fluoro to find the ideal area (low and posterior), wire in, dilate up, and then place the tube.



Nephrostomy on Transplant - They will want you to say that the procedure is contraindicated, it is not. In fact, it is relatively easy to perform compared to a native kidney because of the superficial location of the transplanted kidney.

Complications: Bleeding and urosepsis. The larger the tube, the greater the risk. Bloody urine for 24-48 hours is expected and usually clears after that. Rapid bleeding via urine (from arterial damage) is another story.

Catheter Maintenance: Exchange is required every 2-3 months because of the crystallization of urine in the tube. Some hospitals / departments will do exchanges more frequently than 2 months and that is because of how well this pays... uh I mean they do it for excellent patient care.

Percutaneous Nephrostolithotomy - This is done to remove stones in conjunction with urology. The idea is very similar with a few differences. **The site is sometimes upper pole (instead of lower pole) to make stone access easier.** The tube / hole is bigger and there is more risk of bleeding.

Ureteral Stents- Used for obstruction, leak, prior to complex pelvic surgery etc... They are first line for ureteral injury or leak. They are usually placed retrograde by urology, but can be placed in conjunction with PCN. Don't place them if you are trying to divert past the bladder (duh).

Suprapubic Cystostomy - Done to either (a) acutely decompress the bladder or (b) long term urinary diversion. The best way to do it is with ultrasound in the fluoro suite. You stick low (just above the pubic symphysis) to avoid bowel and peritoneal cavity, and stick in the midline to avoid the inferior epigastrics. Use ultrasound and stick it, confirm position with contrast, wire in and then dilate up. Use a small tube for temporary stuff and a larger tube for more long term stuff. You can always upsize to a foley once the tract is mature.

Renal abscess - Renal abscess is usually secondary to ascending infection or hematogenous spread. The term "perinephric abscess" is used when they perforate into the retroperitoneal space. When they are small (< 3-5cm) they will resolve on their own with the help of IV antibiotics. Indications for aspiration or drainage include a large (> 3-5cm), symptomatic focal fluid collection that does not respond to antibiotic therapy alone. The strategy is to use ultrasound and stick a pig tail catheter in the thing. After a few days if the thing is not completely drained you can address that by upsizing the tube. If you create or notice a urine leak, you'll need to place a PCN. There are really only relative contraindications - bleeding risk etc..., and the procedure is generally well tolerated with a low complication rate.

Renal Biopsy - This can be done for two primary reasons (1) renal failure or (2) cancer biopsy.

Non-Focal: The renal failure workup "non-focal biopsy" is typically done with a 14 - 18 gauge cutting needle, with the patient either prone or on their side (target kidney up). The most obvious testable fact is that **you want tissue from cortex** (lower pole if possible) to maximize the yield of glomeruli on the specimen and minimize complications by avoiding the renal sinus. The complication rate is relatively low, although small AV fistulas and pseudoaneurysms are relatively common (most spontaneously resolve). Some hematuria is expected. In a high risk for bleeding situation a transjugular approach can be done but that requires knowing what you are doing.

Focal: It used to be thought that focal biopsy should NEVER be done because of the dreaded risk of upstaging the lesion and seeding the track. This has been shown to be very rare (<0.01 %). Having said that I think it's still the teaching at least in the setting of pediatric renal masses. This procedure is probably better done with CT. The patient is placed in whatever position is best, but the lateral decubitus with the **lesion side down** is "preferred", as it stabilizes the kidney from respiratory motion, and bowel interposition. Just like with ultrasound, not crossing the renal sinus is the way to go. Just put the needle in the tumor. If it's cystic and solid make sure you hit the solid part. Some texts recommend both fine needle and core biopsy. The core biopsy is going to give a higher yield. A testable pearl is that if lymphoma is thought likely, a dedicated aspirate should be sent for flow cytometry. As with any renal procedure hematuria is expected (not gross - just a little). Renal colic from blood clots is rare.

Renal RFA: Radiofrequency ablation (RFA) is an alternative to partial nephrectomy and laparoscopic nephrectomy. It can be used for benign tumors like AMLs, renal AVMs, and even for RCCs. Angiomyolipomas (AMLs) are treated at 4cm because of the bleeding risk. Sort of a general rule is that things that are superficial you can burn with RFA. Things that are closer to the collecting system it may be better to freeze (cryoablation) to avoid scarring the collecting system and making a stricture. Pyeloperfusion techniques (cold D5W irrigating the ureter) can be done to protect it if you really wanted to RFA. If anyone would ask, RFA has no effect on GFR (it won't lower the GFR).

Things that make you think recurrent/residual disease after therapy. (1) any increase in the size beyond the acute initial increase, (2) areas of "nodular" or "crescentic" enhancement, or (3) a new or enlarging bright T2 signal. There is a paper in AJR (2009) that says that lesions that are <3cm will appear larger in 1-2 months and lesions >3cm do not grow larger - when successfully treated. So, smaller lesions may initially get bigger but after that - any increase in size should be considered tumor recurrence.

Renal Arteriography: You should always do a non-selective aortogram first to see how many arteries feed the kidney, where they are, etc. Sometimes the aortogram will show you an obvious ostial problem which you can then select down on and address. Otherwise, you need to do selective angiography and look at each vessel. **LAO is the projection of choice for looking at the renals.** Sometimes the stenosis is further out, in fact branch artery stenosis is a cause of hypertension in kids.

Angioplasty of Renal Arteries: Used to treat hypertension caused by atherosclerosis (usually ostial) or FMD. Risks include thrombosis, and vessel spasm. Calcium channel blockers can be given to decrease the risk of spasm. Heparin should be on board to reduce thrombosis risk. Most people take daily aspirin the day before and every day after for 6 months, to reduce the risk of restenosis.

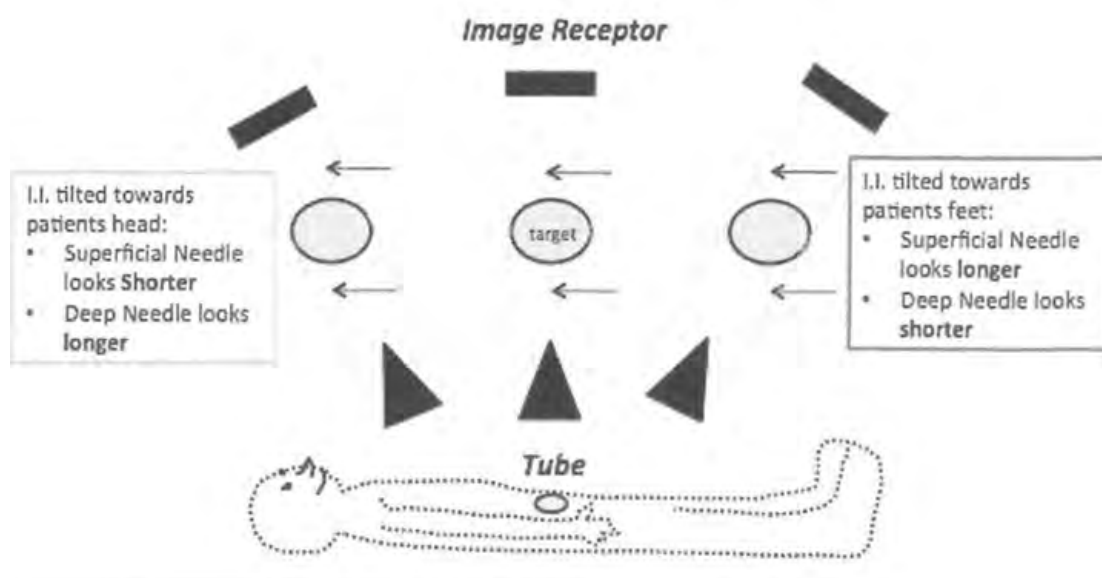
- *Atherosclerosis at the Ostium = Angioplasty + Stent*
- *FMD - usually mid vessel = Angioplasty Alone*

Superficial or Deep? - Understanding Geometry

Sometimes it's difficult to tell if you are superficial or deep to the lesion you are trying to put a needle in under fluoro. You can problem solve by tilting the I.I. towards the patient's head or towards the patient's feet.

If you tilt towards the head, a superficial needle will be shorter but a deep needle will look longer.

If you tilt towards the feet, a superficial needle will be longer but a deep needle will look shorter.

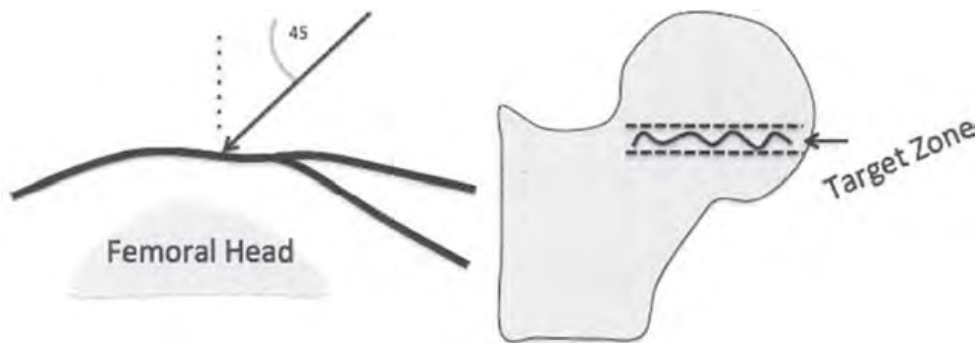


Vascular

Femoral Artery Access - The external iliac becomes the CFA after it gives off the inferior epigastric.

The ideal location is over the femoral head (which gives you something to compress against), distal to the inguinal ligament / epigastric artery and proximal to the common femoral bifurcation.

- * If you stick too high (above inguinal ligament): You risk retroperitoneal bleed
- * If you stick too low, you risk AV Fistula
- * If you stick at the bifurcation: You risk occluding branching vessels with your sheath.



Brachial Access - This can be tricky because holding pressure is often difficult. Even a small hematoma can lead to **medial brachial fascial compartment syndrome** (cold fingers, weakness) - and is a surgical emergency which may require fasciotomy.

Pre Procedure Trivia: Prior to an arterial stick you have to know some anticoagulation trivia. *

- Stop the heparin 2 hours prior to procedure (PTT 1.2x of control or less; normal 25-35 sec)
- INR of 1.5 is the number I'd pick if asked (technically this is in flux)
- Stop Coumadin at least 5-7 days prior (vitamin K 25-50mg IM 4 hours prior, or FFP/ Cryo)
- Platelet count should be > 50K (some texts say 75)
- Stop ASA/Plavix 5 days prior (according to SIR)

Post Procedure Trivia: By the book, you want 15 minutes of compression. You can typically pull a sheath with an ACT of <150-180. Heparin can get turned back on 2 hours post (assuming no complications). Groin check and palpate pulses should be on the post procedure nursing orders.

Wires:

Just some general terminology:

- * 0.039 inch = 1mm
- * 0.035 inch is the usual size for general purposes
- * 0.018 and 0.014 are considered microwires
- * “Glide Wires” are hydrophilic coated wires that allow for easier passage of occlusions, stenosis, small or tortuous vessels

Catheters - general

3 French = 1mm (6 French = 2mm, 9 French = 3mm, etc...)

Diameter in mm = Fr / 3

Important trivia to understand is that the French size is the external diameter of a catheter (not the caliber of the internal drainage canal).

Sheaths are used during cases that require exchange of multiple catheters. The sheath allows you to change your catheters / wires without losing access. They are sized according to the largest catheter they will accommodate.

For example, a 5 French sheath will fit a 5 French Catheter. BUT the outer diameter of the sheath is actually 1.5-2 French larger than the sheath “size.” I think that most people will use 2 - if forced to convert to mm.

- * *Example (1) What is the size of a puncture hole in mm of a 6 French sheath?*

Diameter in mm = Fr / 3

2.7 mm = (6 + 2) / 3

- * *Example (2) What is the size of a puncture hole in mm of a 6 French sheath, placed coaxially into a short access sheath ?*

6 French sheath will need a 8 French inner diameter, which will be a 1 OF outer diameter. $10/3 = 3.3$ mm

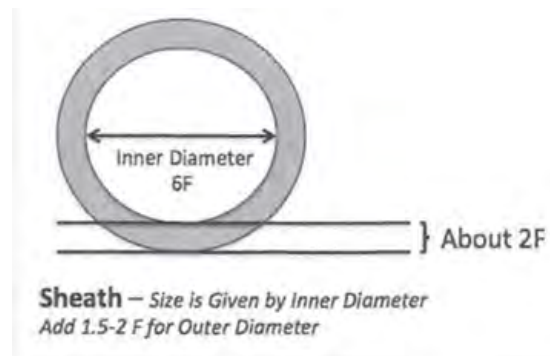
High Yield Points

Catheter Size is given for OUTER LUMEN

Sheath Size is given for the INNER LUMEN

Standard Sizes:

- 6-8F for most routine vascular work
- 6-9F for a Filter (8.5 for Tulip)
- Stent Grafts will need 15F-20F
- Most Abdominal Drains around 12F
- PEGs are 24F



Standard Guide wires are 0.035-0.038 **INCHES** ; 0.038 inches = 3 F. 3F = 1mm. Seriously, watch those units: inches and mm.

Venous Access:

PICC lines: Use the non-dominant arm. The preference is basilic > brachial > cephalic. You don't place these in patients with CRF, on dialysis, or maybe going to be on dialysis.

Central Lines/Port: The right IJ is preferred. External jugular veins can be used.

Subclavian access is contraindicated in patients with a contraindication to PICC lines. Don't place any tunneled lines/ports in septic patients (they get temporary lines).

National Kidney Foundation-Dialysis Outcome Quality Initiative (NKF-KDOQI): Order of preference for access: RIJ > LIJ > REJ > LEJ. "Fistula First Breakthrough Initiative": is the reason you don't place PICCs in dialysis patients.

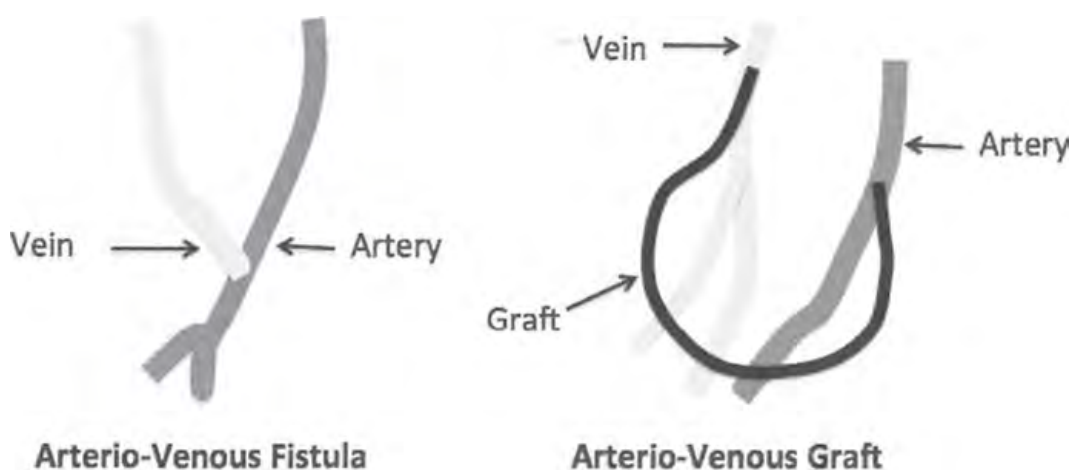
What is the preferred access site for a dialysis catheter? The right IJ is the preferred access, because it is the shortest route to the preferred location (the cavoatrial junction). It will thrombose less than the subclavian (and even if it does, you don't lose drainage from the arm - like you would with a subclavian). Femoral approach is less desirable because the groin is a dirty dirty place.

Dialysis Access / The Dreaded Fistulogram

Types: Generally speaking there are two types; (1) the arterio-venous fistula and (2) the arterio-venous graft.

AVFistula - This is a subcutaneous anastomosis between an artery and adjacent native vein (for example the radial artery to the cephalic vein).

AV Graft - This is also a subcutaneous anastomosis between an artery and adjacent native vein. Except this time the distance between the vessels is bridged with a synthetic tube graft.



What are the pros and cons of each types?

Pros of AV Fistula:

- Lasts Longer & More Durable
- Much less prone to development of neointimal hyperplasia
- Fewer infections

Cons of AV Fistula:

- Needs 3-4 Months to “Mature” (vein to enlarge enough for dialysis)

Pros of AV Graft:

- Ready for use in 2 weeks
- Easier to declot (clot is usually confined to the synthetic graft)

Cons of AV Graft:

- Less overall longevity
- Promotes hyperplasia of the venous intima at or downstream from the graft vein anastomosis, resulting in stenosis and eventual obstruction
- More infections (foreign graft material)

Why do grafts need treated? The primary reason is “slow flows.” It’s important to understand that nephrologists get paid per session of dialysis. If they can do a session in 1 hour, or 4 hours they make the same amount of money. Therefore they want them running fast. So, really “slow-flow” is referring to slow cash flow in the direction of the nephrologist’s pocket. **For the purpose of multiple choice the goal is 600cc/min flow, with an outlet vein > 6mm.** Also remember medicare won’t pay for two treatments within 90 days, so make sure you treat on day 91.

Where is the problem (usually) in grafts? The most common site of obstruction is venous outflow (usually at or just distal to the graft to vein anastomosis). This is usually secondary to intimal hyperplasia.

Where is the problem (usually) in fistulas? It’s more variable - you are less likely to be asked this.

“Steal Syndrome” - The classic story is “cold painful fingers” during dialysis, relieved by manual compression of the fistula. Too much blood going to the fistula leaves the hand ischemic. The issue is usually a stenosis in the native artery distal to the fistula

General Vascular Access Trivia: Remember that PICC lines should not be put in dialysis (or possible dialysis - CKD 4 or 5) patients because they might need that arm for a fistula.

Stenosis Measurements

When stenosis is reported as a percentage (“60% stenosis”), this is referring to the diameter of the artery. The stenosis is determined by comparing the narrowest diameter of the residual lumen to the estimated normal (original) diameter of the lumen at the SAME location.

PTA and Stents

General Tips/Trivia regarding angioplasty: The balloon should be big enough to take out the stenosis and stretch the artery (slightly). The ideal dilation is about 10-15% over the normal artery diameter. Obviously you want the patient anticoagulated, to avoid thrombosis after intimal injury. The typical rule is 1-3 months of anti-platelets (aspirin, clopidogrel) following a stent.

Primary Stenting: This is angioplasty first, then stent placement. You want to optimize your result. Stenting after angioplasty usually gives a better result than just angioplasty alone (with a few exceptions - notable FMD - to which stenting adds very little). An important idea is that a stent can’t do anything a balloon can’t. In other words, the stent won’t open it any more than the balloon will, it just prevents recoil.

Balloon Expanding vs Self Expanding: Stents come in two basic flavors, balloon expandable or self expanding. Location determines the choice. Self expandable stents are good for areas that might get compressed (superficial locations - cervical carotid, or SFA). For more precise deployment the balloon expanding is a good choice (renal ostium).

Nitinol (magic?): Nitinol is said to have a “thermal memory.” It is soft at room temperature, but can become more rigid at body temperature. This is exploited for self-expanding stents.

Drug Alluding Stents - These things have been used for CAD for a while. The purpose of the “drug” is to retard neointimal hyperplasia.

Threatened Limb: Acute limb ischemia can be secondary to thrombotic or embolic events.

Frequent sites for emboli to lodge are the common femoral bifurcation and the popliteal trifurcation. You can also get more distal emboli resulting in the so called “blue toe syndrome.”

As crazy as this may sound to a Radiologist, physical exam is actually used to separate patients into 3 categories: viable, threatened, irreversible.

Know who you can and can't treat

		Capillary Refill	Motor Impairment	Sensory Impairment	Arterial Doppler	Venous Doppler
Viable		Intact	None	None	Audible	Audible
Threatened	Salvageable if treated promptly	Slow	Mild	Mild	Inaudible	Audible
Irreversible	Amputation required	Absent	Rigid	Profound	Inaudible	Inaudible

“Critical Limb Ischemia” - This is described rest pain for two weeks (or ulceration, or gangrene),.

General Idea on Treatment: An important point to realize is that lysis of a clot only re-establishes the baseline (which is likely bad to start with). So after you do lysis consider additional therapy (angioplasty, surgery, stenting, etc...). If there is combined inflow and outflow disease you should treat the inflow first (they just do better).

Surgery vs Thrombolysis: If it has been occluded for < 14 days thrombolysis is superior, if more than 14 days (surgery is superior).

Ankle - Brachial Index (ABI):

Calculation: This is done by dividing the higher of either the dorsalis pedis or posterior tibial systolic pressure (at the ankle) by the higher of either the right or left arm systolic pressure.

What it means: It should normally be 1.0 or higher. ABI is useful for identifying the presence or absence of arterial disease proximal to the ankle. You typically see claudication at 0.5-0.9, and “rest pain” is seen around 0.3.

Ulcer Location Trivia (dinosaur IR guys love this):

- * Medial Ankle = Venous Stasis
- * Dorsum of Foot = Ischemic or Infected ulcer
- * Plantar Surface of Foot = Neurotrophic Ulcer

Who are Rutherford and Fontaine? These are categories and classifications for signs and symptoms of peripheral arterial disease.

False Numbers? Arterial calcifications (common in diabetics) make compression difficult and can lead to a false elevation of the ABI.

Post-Operative Bypass Vocabulary:

- * **Primary Patency** - Uninterrupted patency of the graft with no procedure done on the graft itself (repair of distal vessels, or vessels at either anastomosis does not count as loss of primary patency).
- * **Assisted Primary' Patency** - Patency is never lost, but is maintained by prophylactic interventions stricture angioplasty etc..).
- * **Secondary Patency** - Graft patency is lost, but then restored with intervention (thrombectomy, thrombolysis, etc..).

Varicose Vein Treatment: Just know that “tumescent anesthesia” (lots of diluted subcutaneous lidocaine) is provided for ablation of veins. Veins are ablated using an endoluminal heat source. A contraindication to catheter based vein ablation is DVT (they need those superficial veins).

Post Thrombotic Syndrome (PTS): This is basically pain and stuff (venous ulcers), after a DVT. Risk factors include being old (>65), a more proximal DVT, recurrent or persistent DVT, and being fat. Catheter directed intrathrombus lysis of iliofemoral DVT is done to prevent post thrombotic syndrome. This is not needed as much with femoropopliteal DVT as it will recanalize more frequently and have less severe post thrombotic syndrome.

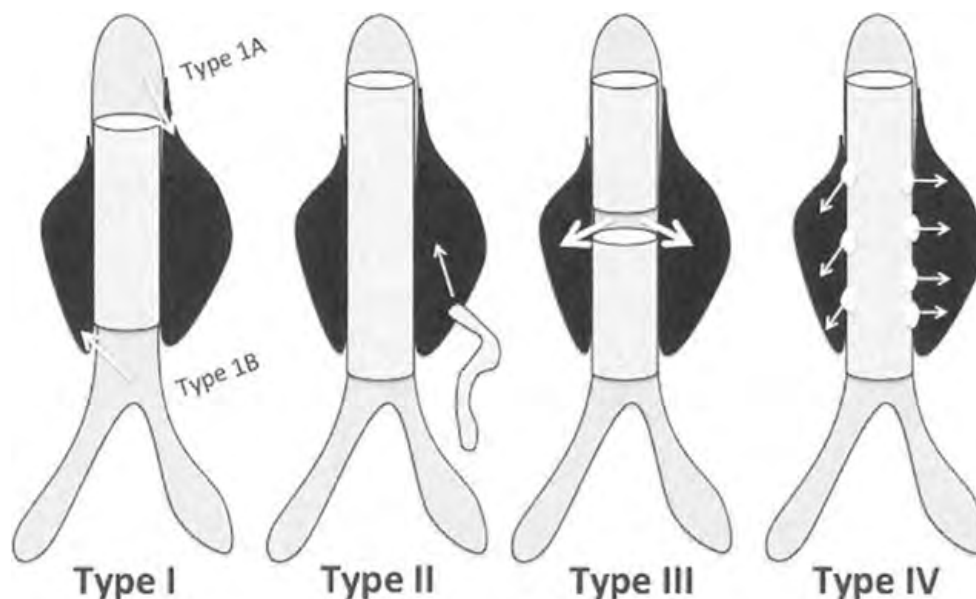
AAA pre/ post Endograft

After an aneurysm has been treated with an endograft, things can still go south. There are 5 described types of endoleaks that lend themselves easily to multiple choice questions.

- * **Type 1** : Leak at the top (A) or the bottom (B) of the graft. They are typically high pressure and require intervention (or the sac will keep growing).
- * **Type 2**: Filling of the sac via a feeder artery. This is the **MOST COMMON** type, and is usually seen after repair of an abdominal aneurysm. The most likely culprits are the IMA or a Lumbar artery. They often spontaneously resolve, but may require treatment. Typically, you follow the sac size and if it grows you treat it.
- * **Type 3** : This is a defect/fracture in the graft. It is usually the result of pieces not overlapping.
- * **Type 4**: This is from porosity of the graft. (“4 is from the Pore”). It’s of historic significance, and doesn’t happen with modern grafts.
- * **Type 5** : This is endotension. It’s not a true leak and maybe due to pulsation of the graft wall. Some people don’t believe in these, but I’ve seen them. They are real.

High Pressure Endoleaks

Type 1 and Type 3 are considered high pressure, because they communicate directly with systemic blood flow.



High Yield Trivia Regarding Endografts vs Open Repair:

- * 30 Day Mortality is LESS for Endovascular Repair
- * Long Term Aneurysm Related Mortality (and total mortality) is the SAME for open vs endovascular repair
- * Graft Related Complications and Re-interventions are HIGHER with Endovascular Repair

Aortic Pathology - Please refer to the vascular chapter for complete discussion of aortic pathology.

Filters

Filters - An IVC filter is used in the following situations: (1) Proven PE while on adequate anticoagulation, (2) Contraindication to anticoagulation with clot in the femoral or iliac veins, (3) Needing to come off anticoagulation - complications. There are a few additional indications that are less firm (basically, we think he/she might get a DVT and we can't anticoagulate).

Vocab:

- * *Permanent Filters:* Doesn't Come Out
- * *Retrievable Filter:* Can Come Out, But Doesn't Have To
- * *Temporary Filter:* Comes out, and has a component sticking outside the body to aide in retrieval

The device is usually placed infrarenal with a few exceptions (see below chart). Why isn't it just place suprarenal? A supra-renal filter has a theoretic increased risk of renal vein thrombosis. There is zero evidence behind this - like most things in medicine.

Indication	Filter Placement	
Pregnancy	Supra-renal	to avoid compression
Clot in the Renals or Gonadals	Supra-renal	get above the clot
Duplicated IVCs	Either bilateral iliac, or supra-renal (above the bifurcation)	
Circumaortic Left Renal	Below the lowest renal	risk of clot by passing filter via the renals

Mega-Cava: If the IVC is less than 28mm, then any filter can be placed. If it's bigger than that, you might need to place a bird's nest type of filter which can be used up to 40mm. You can also just place bilateral iliac filters.

Complications/Risks:

- * *Malposition:* The tip of the filter should be positioned at the level of the renal vein. If it's not, honestly it's not a big deal
- * *Migration:* The filter can migrate to another part of the IVC, the heart, or even the pulmonary outflow tract. If it goes to the heart, you need surgery. If it's just superior, you need to snare it out.
- * *Thrombosis:* Although the incidence of PE is decreased, the **risk of DVT is increased**. Caval thrombosis is also increased, and you should know that clot in the filter is a contraindication to removal (you need to lyse it out, before you remove it).
- * *IVC Perforation:* A strut going through the caval wall is common and doesn't mean anything. However, aortic penetration, ureteral perforation, duodenal perforation, or lumbar vessel laceration can occur (rarely) from a strut hanging out of the cava - this is a bigger problem.
- * *Device Infection:* A relative contraindication to IVC filter placement is bacteremia.

Additional Trivia:

- * A "Gunther Tulip" has a superior end hook for retrieval
- * A "Simon-Nitinol" has a low profile (7F) and can be placed in smaller veins (like an arm vein).
- * All filters are MRI compatible

Misc

Vertebroplasty - There is a paper in the NEJM that says this doesn't work. Having said that, NEJM doesn't like any procedures. They're run by family doctors. They are equally amoral to the person that will do any non-indicated procedure. Regardless of the actual legitimacy, it's a big cash cow and several prominent Radiologists have made their names on it... so it will be tested on as if it's totally legit and without controversy.

Trivia to Know:

- * There is a risk of developing a new vertebral fracture in about 25% of cases. The literature says you should *"counsel patients on the need for additional treatments prior to undergoing vertebroplasty."*
- * The cement can embolize to the lungs.

Lymphangiogram: This is done by first injecting about 0.5cc in between the toes bilaterally. You then wait a half an hour until the blue lymphatic channels are visualized, you then cut down over the lymphatic channels and cannulate with a 27 or 30 gauge lymphangiography needle. An injection with lipiodol is done (maximum 20ml if no leak). You take spot films in a serial fashion until the cistema chyli is opacified. If you inject too much there is a risk of oil pneumonitis.

Skin Dose Changes:

- * 2 Gy- *Early Transient Erythemia*
- * 6 Gy - *Chronic Erythemia*
- * 10 Gy - *Telengiectasia*
- * 13 Gy- *Dry Desquamation*
- * 18 Gy- *Moist Desquamation*

Pseudoaneurysm Treatment: As described in the vascular chapter you can get a pseudoaneurysm after a visit to the cath lab (or other rare causes). A lot of the time, small (<3cm) will undergo spontaneous thrombosis. There are 3 main options for repair: (1) open surgery, (2) direct ultrasound compression, or (3) thrombin injection.

Direct Compression	Direct compression of the neck (if possible avoid compression of the sac). Enough pressure should be applied to stop flow in the neck.	Painful for the patient (and the radiologist), can take 20 mins to an hour.
Thrombin Injection	Needle into apex of cavity - inject 0.5-1.0ml (500-1000 units). Do NOT aspirate blood into syringe - will clot.	<i>Contraindications:</i> Local infection, Rapid Enlargement, Distal Limb Ischemia, Large Neck (risk for propagation), Pseudoaneurysm cavity size < 1cm.
Surgery	May be needed if thrombin injection fails, there is infection, there is tissue breakdown, or the aneurysm neck is too wide.	

Projections with Angio:

Artery of Interest	C Arm Angulation	Misc
Aortic Arch	70 Degrees LAO	“Candy Cane”
Innominate A.	RAO	
Left Subclavian	LAO	
Left Renal	LAO	Same side as renal
Right Renal	RAO	Same side as renal
Left Iliac Bifurcation	RAO	Opposite side common
Right Iliac Bifurcation	LAO	Opposite side common
Right SFA/ Profunda	RAO	Same Side SFA
Left SFA/ Profunda	LAO	Same side SFA

Medications/ Ect:*Anti-Coagulation Issues:*

- * Remember that Platelets Replace Platelets.
- * Heparin: The half life is around 1.5 hours. Protamine Sulfate can be used as a more rapid Heparin Antidote.
- * Coumadin: Vitamin K can be given for Coumadin but that takes a while (25-50mg IM 4 hours prior to procedure), more rapid reversal is done with factors (cryoprecipitate).
- * Remember that patients with “HIT” (Heparin Induced Thrombocytopenia) are at increased risk of clotting - not bleeding. If they need to be anti-coagulated then they should get a thrombin inhibitor instead.
- * The Half Life of a Platelet is 8-12 days

Sedation Related:

- * “Conscious Sedation” is considered “moderate sedation”, and the patient should be able to respond briskly to stimuli (verbal commands, or light touch). No airway intervention should be needed.
- * Flumazenil is the antidote for Versed (Midazolam).
- * Narcan is the antidote for Opioids (Morphine, Fentanyl).

Medication	Mechanism	Trivia
Aspirin	Inhibits thromboxane A ₂ from arachidonic acid by an irreversible acetylation	Irreversible - works the life of the platelet (8-12 days).
Heparin	Binds antithrombin 3	Monitored by PTT. Can be reversed with protamine sulfate
Plavix (Clopidogrel)	Inhibits the binding of ADP to its receptors - leads to inhibition of GP IIb/IIIa	
Coumadin	Inhibits vitamin K dependent factors (2,7,9,10)	Monitored by INR. Delay in onset of activity (8-12 hours). Action can be antagonized by vitamin K - but this takes time (4 hours). For immediate reversal give factors (cryoprecipitate)
Thrombolytic Agents (TPA)	Act directly or indirectly to convert plasminogen to plasmin (cleaves fibrin)	TPA has a very short biologic half life - between 2-10 mins.

Local Anesthesia (Lidocaine)

- Maximum Dose is 4-5mg/kg
- Remember that small doses in the right spot can cause a serious reaction.
 - 150mg in the thecal sac can cause total spine anesthesia and the need for a ventilator.
 - Direct arterial injection can cause immediate seizures.
 - Tinnitus and dizziness are the earliest signs of toxicity.
- Local anesthesia agents have a low potential for allergy - although it can still occur, it's usually a bogus allergy once a real history is taken. Most "allergies" to lidocaine are actually vaso-vagal, or other CV side effects from epinephrine mixed with lidocaine
- There are elaborate mechanisms for testing for a true allergy, or reaction to methylparaben (a preservative).
- So what if the allergy is real? or you can't prove it's false? - Some texts describe using an antihistamine such as diphenhydramine (which can have anesthetic properties).

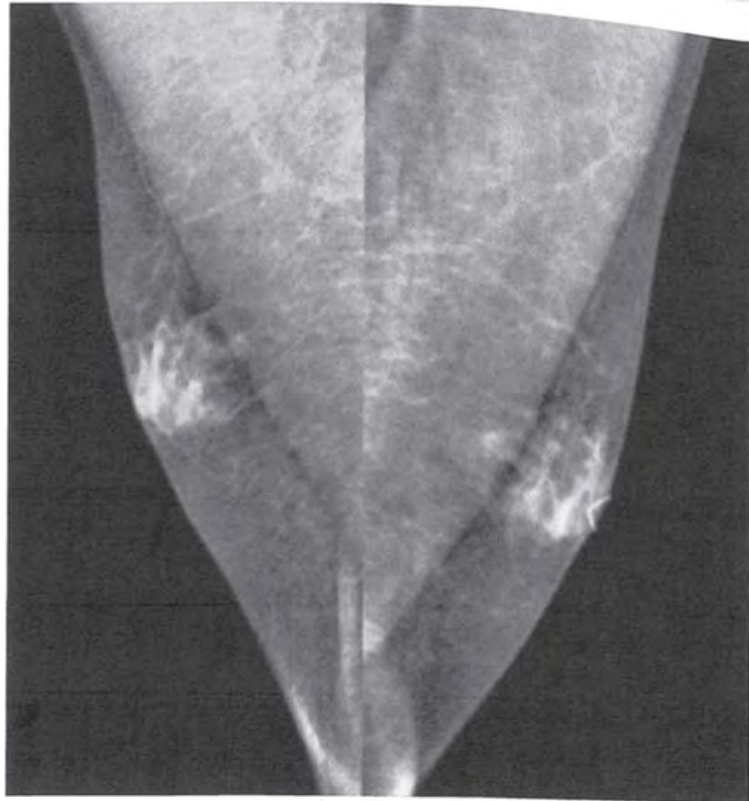
Green, Steven M., Steven G. Rothrock, and Julie Gorchynski. "Validation of diphenhydramine as a dermal local anesthetic." Annals of emergency medicine 23.6 (1994): 1284-1289.

	Indications	Contraindications
Angiography	Numerous; usually diagnosis of and treatment vascular disease	Only one absolute which is an unstable patient with multisystem dysfunction (unless angio is life saving). There are numerous relatives including, inability to lay flat, uncooperative patient, and connective tissue diseases
Ascending Venography	Diagnosis of DVT, Evaluate Venous malformation or tumor encasement.	Contrast Reaction Pregnancy Severely compromised cardiopulmonary status
Descending Venography	Evaluation of post-thrombotic syndrome; valvular incompetence and damage following DVT	
Venography (Non-inclusive)	Thoracic Outlet Syndrome, Venous Access, Pacer Placement, Eval for fistula	
IVC Filter	Can't get anticoagulation, Failed anticoagulation (clot progression), Massive PE requiring lysis, Chronic PE treated with thromboendarterectomy. Trauma high risk DVT	Total thrombosis of IVC IVC too big or too small <i>*Sepsis is NOT a contraindication, including septic thrombophlebitis</i>
Fistulography	Making the nephrologist money ("slow flows" they call it).	Absolute: Right to left cardiopulmonary shunt, Uncorrectable coagulopathy, fistula infection. Relative is significant cardiopulmonary disease (a declot invariably causes PE)

	Indications	Contraindications
TIPS	Variceal bleeding refractory to endoscopy. Refractory ascites.	Absolute: Heart Failure (especially right heart failure). Severe encephalopathy. Rapidly progressing liver failure.
Percutaneous Transhepatic Cholangiography (PTC)	Performed prior to percutaneous biliary interventions, Choledochojejunostomy patients (liver transplant) with suspected obstruction	Absolute: Uncorrectable Coagulopathy, Plavix or other anti-platelet agent Relative: Large Volume Ascites (consider para and left sided approach)
Percutaneous Biliary Drainage	Basically CBD obstruction (with failed ERCP), cholangitis, bile duct injury/leak.	No absolute contraindications Relative: Large Volume Ascites (consider para and left sided approach), Coagulopathy
Percutaneous Cholangiostomy	Cholecystitis in patients who are not surgical candidates, Unexplained sepsis when other sources excluded, Access to biliary tree required and other methods failed	No absolute contraindications Relative: Large Volume Ascites (consider para and left sided approach), Coagulopathy
Percutaneous Nephrostomy	Obstructive Uropathy (Not hydronephrosis), Urinary diversion (leak, fistula), Access for percutaneous intervention	Uncorrectable coagulopathy, Contrast Reactions

10 Breast

Prometheus Lionhart, M.D.



Obviously the amount of pathology in breast imaging is extremely limited. Additionally, most of it will not project well on the computer monitors in most (all) testing centers. As a result, a lot of the eye tests that make practical mammography difficult are unlikely to be shown on the test.

High Yield Trivia

- BI-RADs Terminology
- Think About Management / Next Step Style Questions
- The Male Breast
- Post Operative
- MQSA

Introduction:

Recent large heavily powered studies have brought into question the practice of screening mammograms. I highly recommend you read these “*despicable*” papers, but please wait till after the CORE exam, because the people who write multiple choice questions about mammography are definitely not the same people who wrote these papers. For the purpose of multiple choice tests, screening mammography saves lots of lives, you should buy pink ribbons, and low grade DCIS needs a surgical consult.

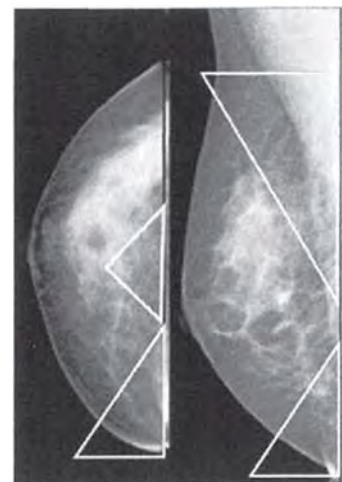
Bleyer, Archie, and H. Gilbert Welch. "Effect of three decades of screening mammography on breast-cancer incidence." New England Journal of Medicine 367.21 (2012): 1998-2005.

Miller, Anthony B., et al. "Twenty five year follow-up for breast cancer incidence and mortality of the Canadian National Breast Screening Study: randomised screening trial." BMJ: British Medical Journal 348 (2014).

Anatomy:

Nipple: The nipple is a circular smooth muscle that overlies the 4th intercostal space. There are typically 5-10 ductal openings. *Inversion* is when the nipple invaginates into the breast. *Retraction* is when the nipple is pulled back slightly. They can both be normal if chronic. If they are new, it should make you think about underlying cancers causing distortion. The nipple is suppose to be in profile, so you don't call it a mass. The areola will darken normally with puberty and parity. Nipple enhancement on contrast enhancement breast MRI is normal, don't call it pagets.

Fibroglandular Tissue: The breast mound is fibrous tissue with fat, ducts, and glands laying on top of the anterior chest wall. The axillary extension is called the “*tail of Spence*” The upper outer quadrant is more densely populated with fibroglandular tissue, which is why most breast cancers start there. There is usually no dense tissue in the medial/ inferior breast and retroglandular regions. These are considered “danger zones” and are often where the cancer hides.

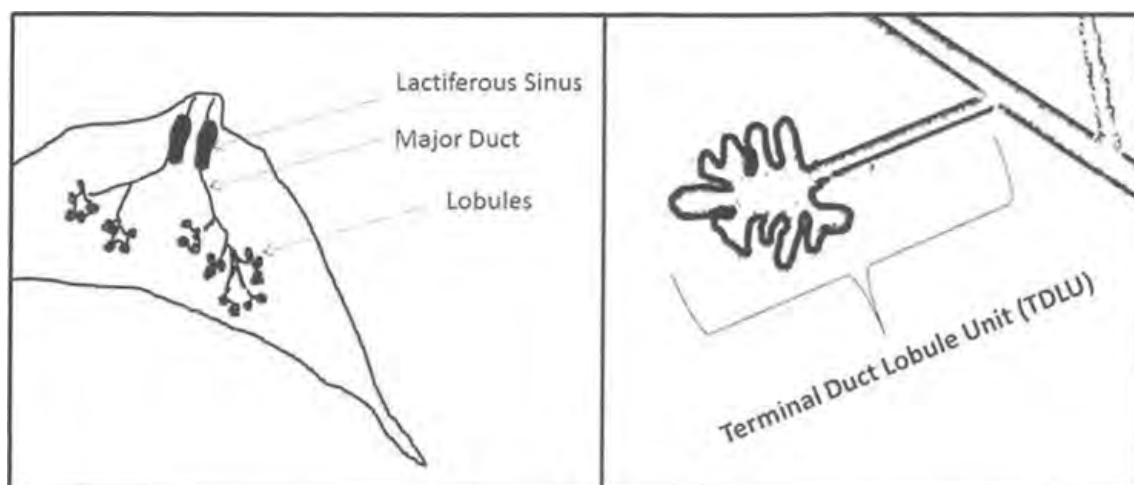


Danger Zones - where there is usually no dense fibroglandular tissue

Cooper Ligaments: These are thin sheets of fascia that holds the breasts up. They are the tiny white lines on mammography and the echogenic lines on US. Straightening and tethering of the ligaments manifests as “architectural distortion” which occurs in the setting of surgical scars, radial scars, and IDC.

Breast Asymmetry: This is common and normal (usually), as long as there are no other findings (lumps, bumps, skin thickening, etc..). *For multiple choice, an asymmetric breast should make you think about the "shrinking breast" of invasive lobular breast cancer.* If the size difference is new or the parenchyma looks asymmetrically dense think cancer.

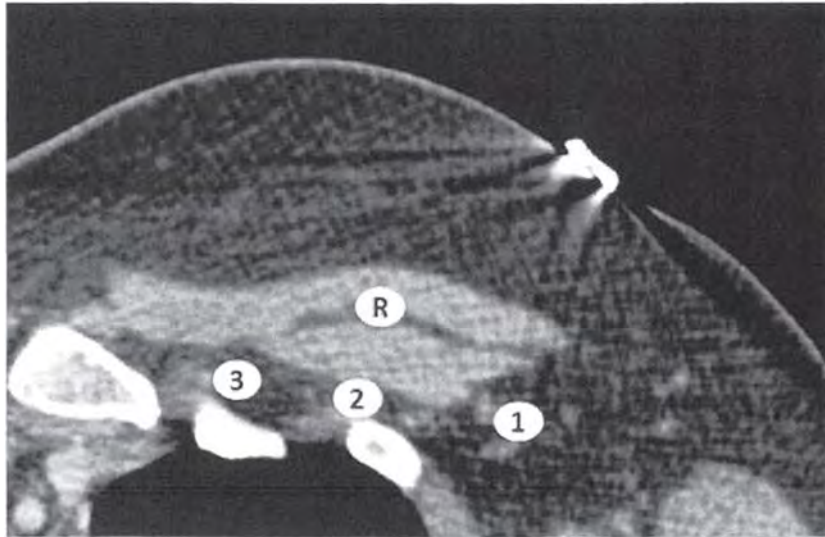
Lobules: The lobules are the flower shaped milk makers of the breast. The terminal duct and lobule are referred to as a “terminal duct lobule unit” or TDLU. This is where most breast cancers start.



Ducts: The ductal system branches like the roots or branches of a tree. The branches overlap wide areas and are not cleanly segmented like slices of pie. The calcifications that appear to follow the duct (“linear or segmental”) are the ones you should worry about cancer with.

Lactiferous Sinus: Milk from the lobules drains into the major duct under the nipple. The dilated portion of the major duct is sometimes called the lactiferous sinus. This thing is normal (not a mass).

Blood Supply / Lymphatic Drainage: The majority (60%) of blood flow to the breast is via the internal mammary. The rest goes to the lateral thoracic and intercostal perforators. Nearly all (97%) of lymph drains to the axilla. The remaining 3% goes to the internal mammary nodes.



Axillary Lymph Node Levels:

Level 1: Lateral to Pec Minor

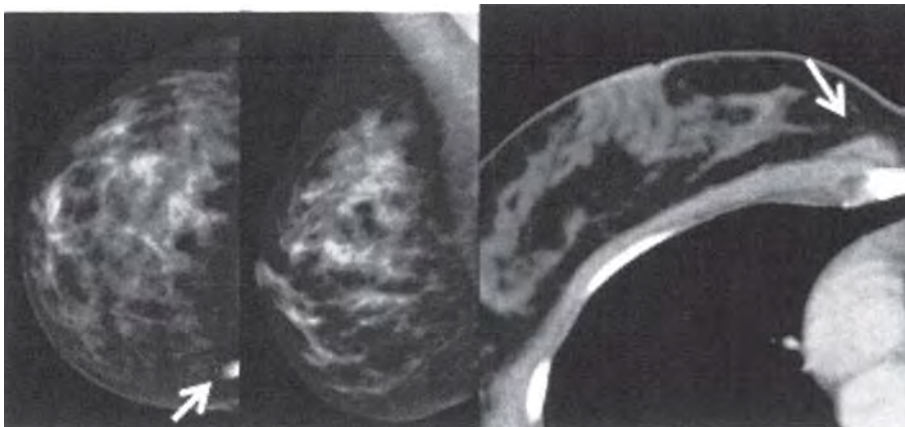
Level 2: Under Pec Minor

Level 3: Medial to Pec Minor

Rotter Node: Between Pec Major and Minor

Metastasis to the Internal Mammary Nodes: If you can see them on ultrasound they are abnormal. Isolated mets to these nodes is not a common situation (maybe 3%). When you do see it happen it's from a medial cancer.

Sternalis Muscle: This is an Aunt Minnie. It's a non-functional muscle next to the sternum that can simulate a mass. It is **ONLY SEEN ON THE CC VIEW**. It's usually unilateral more than half of the time. Handling this in real life is all about the old gold. Find that thing on the priors (even better is a CT), CC only, **never on the MLO**.



Sternalis - Only on CC, Never on MLO

Breast Development / Physiology: The “milk streak” is the embryologic buzzword to explain the location of the normal breast and location of ectopic breast tissue. Just know that the **most common location for ectopic breast tissue is in the axilla** (second most common is the inframammary fold). Extra nipples are most commonly in the same locations (but can be anywhere along the “milk streak”). At birth both males and females can have breast enlargement and produce milk (maternal hormones). As girls enter puberty their ducts elongate and branch (estrogen effects), then their lobules proliferate (progesterone effects). If you biopsy a breast bud (why would you do that?) you can damage it and affect breast development.... and then get sued.

- *Follicular Phase (day 7-14):* Estrogen Dominates. Best time to have both mammogram and MRI.
- *Luteal Phase (day 15-30):* Progesterone Dominates. This is when you get some breast tenderness (max at day 28-30). Breast density increases slightly.
- *Pregnancy:* Tubes and Duct Proliferate. The breast gets a lot denser (more hypoechoic on US), and ultrasound may be your best bet if you have a mass.
- *Perimenopausal:* Shortening of the follicular phase means the breast gets more progesterone exposure. More progesterone exposure means more breast pain, more fibrocystic change, more breast cyst formation.
- *Menopause:* Lobules go down. Ducts stay but may become ectatic. Fibroadenomas will degenerate (they like estrogen), and get their “popcorn” calcifications. Secretory calcifications will develop (*but not for 15-20 years post menopause).
- *Hormone Replacement Therapy:* Breast get more dense (even more so estrogen-progesterone combos). Breast pain can occur, typically peaking the first year. Fibroadenoma (who like to drink estrogen) can grow.

High Yield Trivia Regarding Breast Anatomy / Physiology

- The nipple can enhance with contrast on MRI. This is normal (not Pagets).
- Most cancers occur in the upper outer quadrant.
- Most cancers start in the terminal duct lobule unit (TDLU).
- Majority (60%) of blood flow is via the internal mammary.
- Mets to the Internal Mammary Nodes are uncommon (3%) - seen in medial cancers.
- Axillary Node Levels (3, 2, 1 - medial to lateral -)
- Stemalis is usually unilateral, and only on the CC, NEVER on MLO.
- Breast Tenderness is max around day 27-30.
- Mammography and MRI are best performed in the follicular phase (days 5-10).
- Don't Biopsy a prepubescent breast - you can affect breast development
- Perimenopause (50s) is the peak time for breast pain, cyst formation
- Fibroadenomas will degenerate (buzzword popcorn calcification) in menopause

What You Know About Lactation?

key by the three when I chirp shawty chirp back

Density: As mentioned above the breast gets a lot denser in the 3rd trimester. Mammograms might be worthless, and ultrasound could be your only hope.

Density Trick: Pituitary Prolactinoma, or meds (classically antipsychotics) can create a similar bilateral increased density.

Biopsy: You can biopsy a breast that is getting ready to lactate / lactating - you just need to know there is **the risk of creating a milk fistula**. If you make one, they will have to stop breast feeding to stop the fistula. The fistula can get infected, but that's not very common.

Galactocele: This is one of those "benign fat containing lesions" that you can BR-2. This is typically seen on cessation of lactation. The location is typically sub-areolar. The appearance is variable, but can have an **Aunt Minnie look with a fat-fluid level**. It's possible to breast abscess these things up.

Lactating Adenoma: These things look like fibroadenomas, and may actually be a charged up fibroadenoma (they like to drink estrogen). Usually these are **multiple**. If you get pressed on follow up recommendation for these I would say 4-6 months post partum, post delivery or after cessation of lactation -via ultrasound. They usually **rapidly regress after you stop lactation**.

Risk

Estrogen Related: The more exposure to estrogen the higher your risk of breast cancer will be. Anything that prolongs this exposure is said to increase risk. For example, an early age to begin menstruating or a late age to have menopause. Hormone replacement therapy with estrogen alone obviously increases exposure.

Early maturation of lobules, which can be achieved by getting pregnant young, reduces your risk. Being fat increases estrogen exposure (more aromatase = more estrogen). Being a drunk increases estrogen exposure - via messing with its normal breakdown in the liver.

Estrogen Related Risks

Early Menstruation
Late Menopause
Late age of first pregnancy / or no kids.
Being Fat
Being a Drunk
Hormone Replacement (with estrogen)

High Risk Lesions: Any of the high risk lesions (ADH, ALD, LCIS, Radial Scar, Papilloma) are associated with an increased risk. These are discussed more in detail later in the chapter.

Density: Density is considered a “medium risk,” and is “dose dependent” with the denser you are the more risk you have.

Chest Wall Radiation: Chest wall radiation (usually seen in lymphoma patients) is a big risk, especially at a young age. The risk is supposed to peak around 15 years post treatment. **If the child had more than 20Gy to the chest she is going to qualify for an annual screening MRI - at age 25 or 8 year post exposure (whichever is later).**

Relatives with Cancer: A first degree relative with breast cancer increases your lifetime risk from 8% to 13%. Two first degree relatives increases your risk to 21%.

Actual Mutations:

BRCA1	Chromosome 17. More common than type 2. Increased risk for breast, ovary, and various GI cancers.
BRCA2	Chromosome 13. Male carriers have a higher risk with 2. Increased risk for breast, ovary, and various GI cancers.
Li Fraumeni	Their p53 does work, and they are high risk for all kinds of rare cancers.
Cowden Syndrome	Risk for breast cancer, follicular thyroid cancer, endometrial cancer, and Lhermitte-Duclos (a brain hamartoma).
Bannayan-Riley Ruvalcaba	Associated with developmental disorders at a young age.
NF-1	“Moderate Risk” of breast cancer

Breast Cancer Risk Models:

There are several risk models, which have pros/cons and differences. I apologize in advance for even suggesting you learn about these.... but it just seems testable to me. I'm sorry...

Gail Model	Oldest and most validated breast cancer risk model	Focuses on person risk factors, biopsy of ADH, and family history	Doesn't use genetics (it's too old school). Only validated in African Americans.
Claus, BODICEA, and BRCApro		Focus on genetics	Does NOT include personal risk or breast related risk factors.
Tyrer-Cuzick	<i>"Most Comprehensive"</i>	Includes Personal Risk, Biopsy with ADH or LCIS, Family History	Does NOT include breast density.

High Yield Take Home Points Regarding Risk:

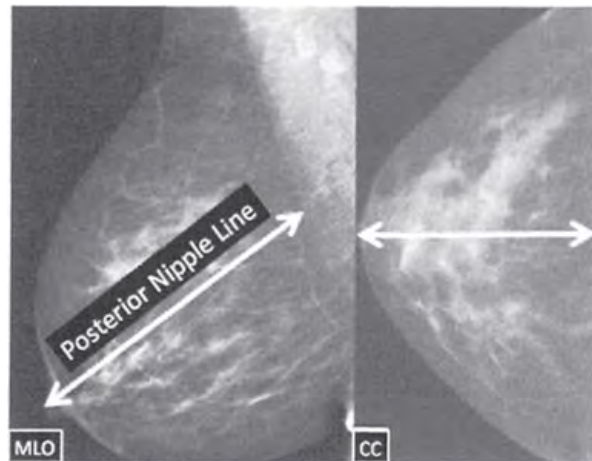
- Anything that gets you more estrogen increases your risk
- * BRCA 1 is more common than BRCA 2 (in women).
- * Men with BRCA 2 get more cancer than men with BRCA 1.
- Breast Density is an independent risk factor (denser the breast, the more the risk)
- * 20Gy of Radiation to your chest as a kid buys you a screening MRI - at 25 or 8 years after exposure (**whichever is longer*)
- * Cowden Syndrome - Bowel Hamartoma, Follicular Thyroid Cancer, Lhermitte-Duclos, and Breast Cancer
- All current risk models underestimate life time risk.
- Tyrer Cuzick is the most comprehensive risk model, but does not include breast density.
- * Exercise (*probably more like not being fat*) reduces the risk of breast cancer
- Tamoxifen and Raloxifene (SERMs) reduce breast cancer incidence of ER/PR

Technique

Is the technique adequate?

The **Posterior Nipple Line** - is drawn on the MLO from the nipple to the chest wall. You need to touch pectoralis muscle to be adequate.

Then on CC, you draw a line from the nipple back towards the chest wall. To be adequate **you must be within 1 cm of the length of the posterior nipple line.**



So other points and trivia:

- * Ideally, the inframammary fold should be visualized
- * “Camel Nose” is the buzzword used to describe a breast on MLO that has not be pulled “up and out” by the tech
- * The nipple should be in profile in one of two views (to avoid missing the subareolar cancer).
- * Relaxed pectoralis muscles are preferred (concave, instead of convex) - showing more breast tissue.

When do you get a LMO view ?

The MLO is the standard, but sometimes you see a LMO. The answer is women with **kyphosis or pectus excavatum**. Or to **avoid a medial pacemaker or line**.

MLO View Trivia: The MLO view contains the most breast tissue of all the possible views

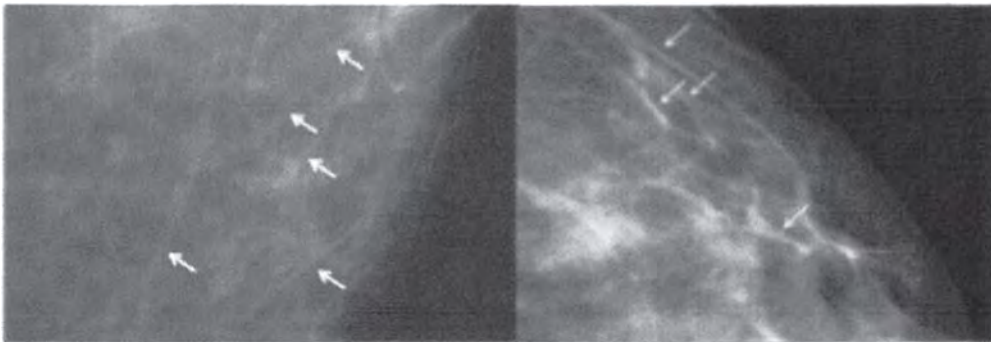
When using Spot Compression Views: A big point is the recommendation to **leave the collimator open**, giving you a larger field of view, and helping to ensure that you got what you wanted to get. Small paddles give you better focal compression. Large paddles allow for good visualization of land marks.

When using Magnification Views: A CC and ML (true lateral) are obtained. You **get a ML (as opposed to a MLO) to help catch milk of calcium**.

When using a True Lateral View ML vs LM: Using a true lateral is useful for localizing things seen on a single view only (the CC). A trick I use is whatever I said on the screener, is the last letter I'd use on the call backs. In other words, if it's Lateral on the screener you want an ML on the diagnostic. If it's Medial on the screener then you want a LM on the diagnostic. The reason is that you are moving it closer to the receptor. **If you see the area of interest on the MLO only (not the CC), you should pick ML - because most (70%) of breast cancers occur laterally.** — This would make a good multiple choice question.

Artifacts:

Blur: Can be from breathing or inadequate compression (typically along the inferior breast on the MLO). It can be tricky to pick up. The strategy I like to use, is to look at the Cooper Ligaments - they should be thin white lines in the fat. If they are thick or fuzzy - it is probably blur (or edema). If there is skin thickening think edema.



Blur: Coopers too thick for normal skin

Grid Lines: Basically mammograms always use a grid (unless it's a mag view). That would make a good multiple choice question actually. No grid on mag views. So, the grid works by moving really fast, and only keeping x-rays that move straight in. You see blur in 3 scenarios (1) patient moved, (2) exposure was too long, (3) exposure was too short.

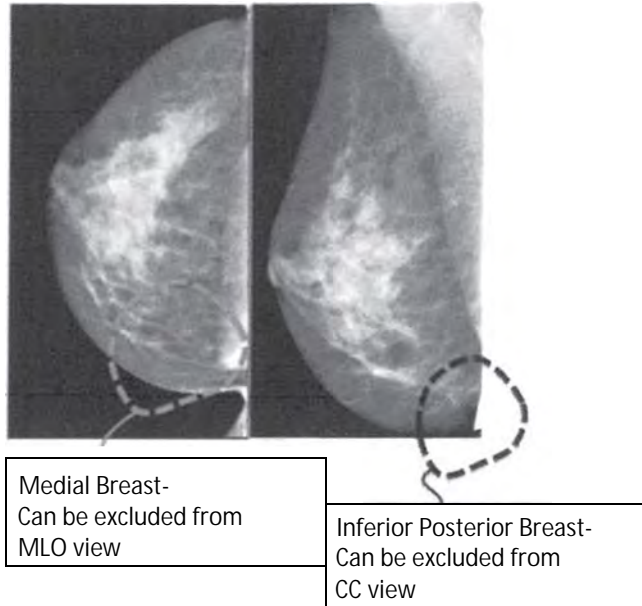


Grid Lines: 3 Examples – horizontal lines

General Tips on Screening Mammograms

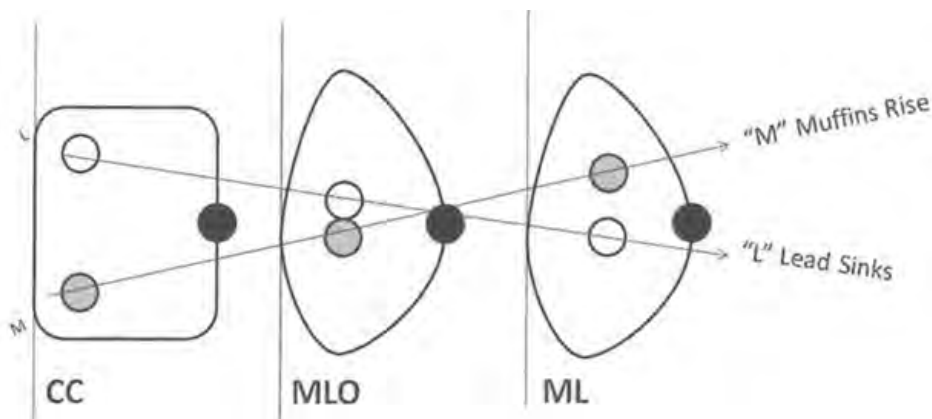
You are trying to find **3-8 cancers per 1000 mammograms** - as demanded by the various regulating bodies

Be aware that certain areas can sometimes only be seen on a single view. For example, the **medial breast on a CC may not be seen on MLO**, and the **Inferior Posterior Breast on MLO may be excluded from the CC**. That makes these areas “high risk” for missing a cancer.



It's recommended to look at mammograms from 2 years prior (if available) for comparison. Makes it a little easier to see early changes.

Localizing a lesion: This is a very basic skill, but if you had absolutely no interest in mammography or just terrible training a refresher might be useful as this is applicable to multiple choice tests. A lesion that is medial on the CC film, will become more superior on the MLO, and even more superior on the ML. The opposite is true of lateral lesions which become more inferior. The popular mnemonic is *“Lead Sinks, and Muffins Rise”* — L for lateral, and M for medial.

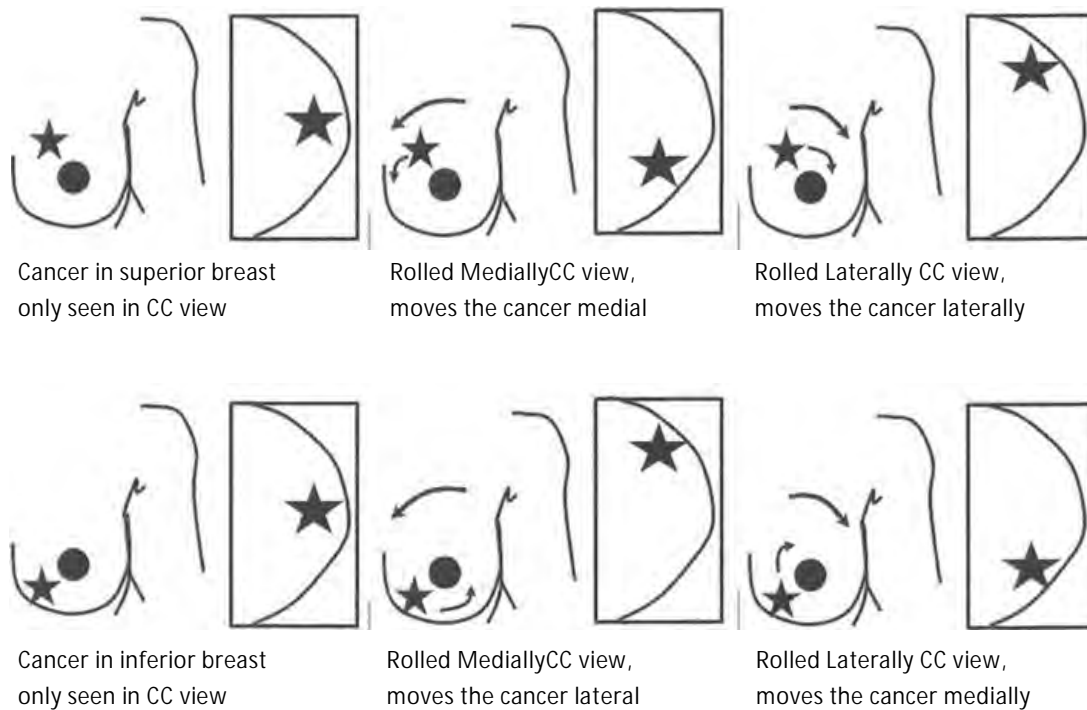


Localizing a lesion (only seen in the CC view): Sometimes you can only see the finding in the CC view. If you want to further characterize it with ultrasound, figuring out if it's in the superior or inferior breast could be very helpful. One method for doing this is a "rolled CC view."

Rolled CC View: This works by positioning the breast for a CC view, but prior to placing the breast in compression you rotate the breast either medial or lateral along the axis of the nipple.

- If you roll the breast medial; a superior tumor will move medial, an inferior tumor will move lateral.
- If you roll the breast lateral; a superior tumor will move lateral, an inferior tumor will move medial.

In other words, **superior tumors move in the direction you roll** and inferior tumors move in the opposite direction you roll.



BI-RADS

BI-RADS is an acronym for Breast Imaging-Reporting and Data System. It was developed by the ACR to keep everyone on the same page, in a similar way the DSM was developed for psych. You can't have people just calling stuff "breast nodules".

* *BI-RADS Assessment Categories:*

- 0: Incomplete
- 1: Negative
- 2: Benign finding(s)
- 3: Probably benign
- 4: Suspicious abnormality
 - * Some people use 4a (low suspicion), 4b (intermediate suspicion), and 4c (moderate suspicion).
- 5: Highly suggestive of malignancy
- 6: Known biopsy - proven malignancy

BI-RADS 0: This is your incomplete workup. They come in for a screener, you find something suspicious. You give it a BI-RADS 0, and bring them back for spots, mags, or ultrasound. You would also BI-RADS 0 anything that required a technical repeat.

BI-RADS 1: It's normal

BI-RADS 2: Benign findings. Examples would be cysts, secretory calcifications, fat containing lesions such as oil cysts, lipomas, galactoceles and mixed-density hamartomas.

- * *Multiple bilateral well circumscribed similar appearing masses* - This is BR-2 unless one is growing or different than the rest. The general rule is to not ultrasound these things unless one is palpable.
- * *Multiple Foci* - This MRI finding, is also a classic BR2.

BI-RADS 3: A key point is that **BR-3 by definition means it has less than 2% chance of being cancer.** This is often a confusing topic. You can only use BR3 on a baseline. You can't call anything BR3 that is new. The typical BR3 scenario is; 45 year old comes in for screening and has a focal asymmetry. She gets called back for diagnostic work up with spots and ultrasound. She is found to have mass with imaging features classic for fibroadenoma. This can get a BR-3, and be followed (some places follow for 2 years, in 6 month intervals). Any change over that time ups it to BR-4 and it gets a biopsy.

Things you can BR-3:

- * *Finding consistent with fibroadenoma*
 - * *Focal asymmetry that looks like breast tissue (becomes less dense on compression).*
 - * *Grouped or Clustered Round Calcifications*
- * *What if it's palpable?* This is a controversial topic. Classic teaching is that palpable lesions can not be BR3. However, recent papers have shown that palpable lesions consistent with fibroadenoma are less than 2% chance of cancer. Some people think the new BI-RADS will change this rule. For the purpose of the CORE exam you can BR-3 a palpable finding, but I really doubt they will paint you into a corner.

BI-RADS 4: This is **defined as having a 2-95% chance of malignancy**. Some people will subdivide *this into 4A, 4B, 4C depending on the level of suspicion*. Ultimately you are going to biopsy it, and be prepared to accept a benign result.

BI-RADS 5: This is defined as **> 95% chance of malignancy**. When you give a BR-5, you are saying to the pathologist “if you give me a benign result, I’ll have to recommend surgical biopsy. ” In other words, you **can’t accept benign with a BR-5**.

BI-RADS 6: This is path proven cancer.

BI-RADS Terminology

In addition to the “0-6” babysitting, the various regulating bodies have decided there are only a few words they will trust you with, depending on what modality you are using.

Plain Mammography:

“Mass” - This is a space occupying lesion seen in two different projections

“Focal Asymmetry” - This is a density (only seen in one view), that may or may not be a mass, and is often a term used in screeners for BR-0 prior to call back. *

- *Global Asymmetry* - “greater volume of breast tissue than the contralateral side”, around one quadrants worth (or more). It’s gonna get a call back, and then BR-2’d on a baseline.
- *Focal Asymmetry* - As above, seen in one view only. Not for sure a mass.
- *Developing Asymmetry* - Wasn’t there before, now is.

Describing the mass: You need to cover (1) Shape, (2) Margin, (3) Density

- (1) *Shape:* Round, Oval, Lobular, Irregular
- (2) *Margin:* Circumscribed, Microlobulated, Obscured, Indistinct, Spiculated
- (3) *Density* (relative to breast parenchyma: Fat Density (radiolucent), Hypodense, Isodense, Hyperdense

Ultrasound:

Describing the mass: You need to cover : (1) Shape, (2) Orientation, (3) Boundary, (4) Echo pattern, (5) Posterior acoustic features

- (1) *Shape:* Round ,Oval, Irregular (not round or oval)
- (2) *Orientation:* Parallel (wider than tall), Anti-Parallel (taller than wide)
- (3) *Margin:* Circumscribed, Indistinct, Angular, Microlobulated, Spiculated
- (4) *Boundary:* Abrupt, Echogenic Halo (interface between mass and surrounding tissue is bridged by an echogenic zone)
- (5) *Echo Pattern:* Anechoic, Hyperechoic, Hypoechoic, Isoechoic, or Complex (*both anechoic and echogenic components*)
- (6) *Posterior Features:* None, Enhancement, Shadowing

MRI

There has recently been a vocabulary change on the Lexicon, and I'm not certain if this is after the CORE was written, so I'm going to present both.

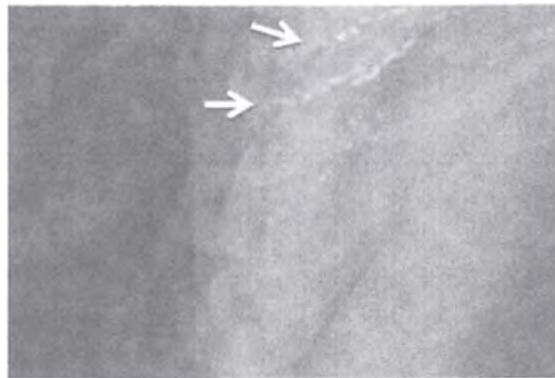
Masses	
Irregular	This is now a combined shape/margin descriptor
Oval	Lobulated is no longer a shape term
Circumscribed	Smooth is no longer a margin term
T2 Signal	- This is new feature
NMLE	
Linear and Linear Branching	- This replaces ductal
Clustered Ring	- This is a a new term
Reticular & Dendritic	Removed because no one uses those
T2 Signal	- This is new feature

Calcifications

Calcifications can be an early sign of breast cancer. “The earliest sign” actually, according to some. Calcifications basically come in three flavors: (1) artifact, (2) benign, and (3) suspicious.

Artifact:

Deodorant: High density material seen in the axilla is the typical appearance. Another trick is to show a speck of high density material that doesn't change position on different views (inferring that it's on the image receptor).



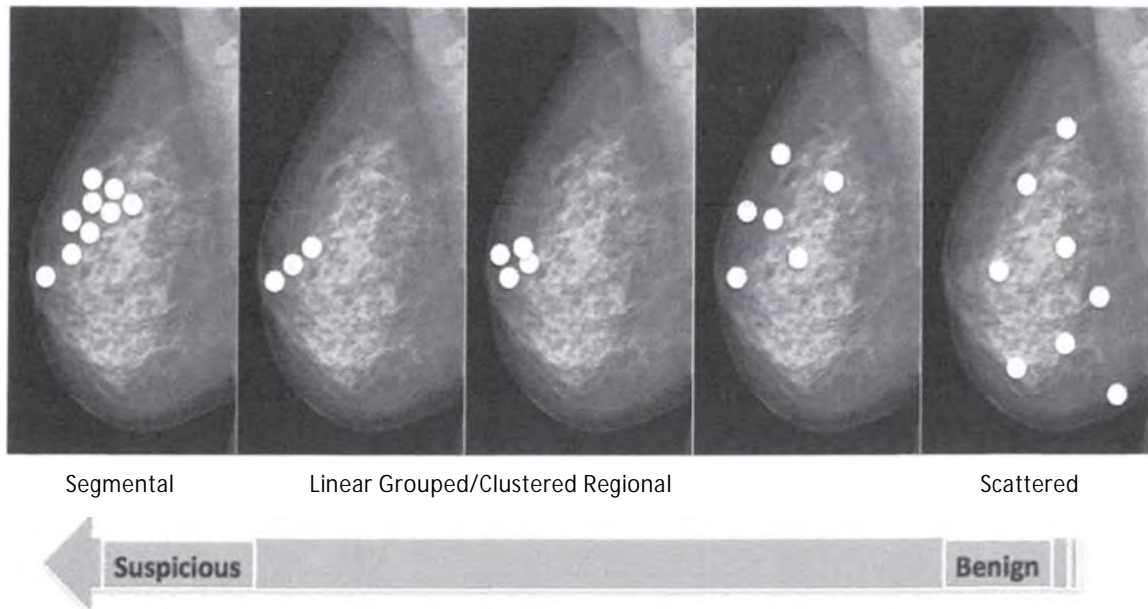
Deodorant Artifact

Zinc Oxide: This is in an ointment old ladies like to put on their floppy sweaty breasts. It can collect on moles and mimic calcifications. If it disappears on the follow up it was probably this (or another dermal artifact).

Metallic Artifact: It's possible for the electrocautery device to leave small metallic fragments in the breast. These will be very dense (metal is denser than calcium). It will also be adjacent to a scar.

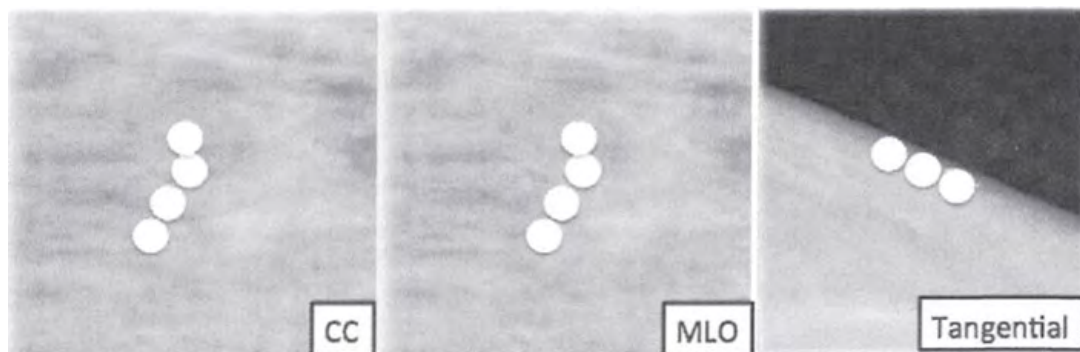
Benign vs Suspicious:

The distinction between benign and suspicious is made based on morphology and distribution (those BI-RADS descriptors). Since most breast cancers start in the ducts (a single duct in most cases), a linear or segmental distribution is the most concerning. The opposite of this would be bilateral scattered calcifications.



Benign:

Dermal Calcifications: These are found anywhere women sweat (folds, cleavage, axilla). Just think folds. They are often grouped like the paw of a bear, or the foot of a baby. The trick here is that these **stay in the same place on CC, and MLO view**. This is the so called **“tattoo sign.”** If you are asked to confirm these are dermal calcs, I’d ask for a “tangential view.”

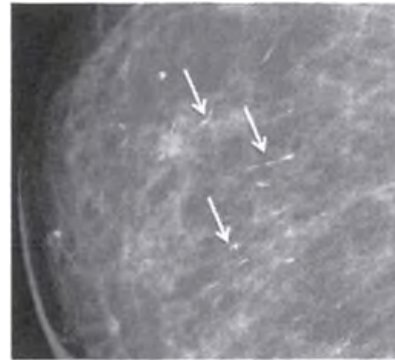


Dermal Calcifications

Vascular Calcifications: These are parallel linear calcifications. It's usually obvious, but not always.

Popcorn Calcifications: This is an immediate buzzword for degenerating fibroadenoma. The typical look is they begin around the periphery and slowly coalesce over subsequent images.

Secretory (Rod-Like) Calcifications: These are big, easily seen, and point toward the nipple. They are typically bilateral. The buzzword is "*cigar shaped with a lucent center.*" Another buzzword is "*dashes but no dots.*" The buzz age is "*10-20 years after menopause.*" **Don't be an idiot and call these in a premenopausal patient,** they happen because the duct has involuted.



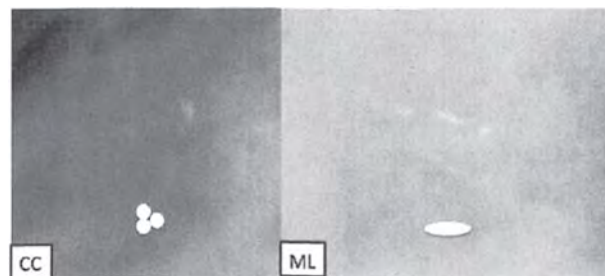
Secretory Calcifications

Eggshell Calcifications: "Fat necrosis" I call them. It can be from any kind of trauma (surgical, or accident - play ground related). If they are really massive you may see the word "**liponecrosis macrocystica.**" As I've mentioned many times in this book, anything that sound Latin or French is high yield for multiple choice. "*Lucent Centered*" is a buzzword.

Dystrophic Calcifications: These are also seen after radiation, trauma, or surgery. These are usually big. The buzzword is "*irregular in shape.*" They can also have a lucent center.

Milk of Calcium: This has a very characteristic look, and because of that questions can only be asked in one of two ways: (1) what is it? - shown as CC then ML, (2) what is it due to ?

- (1) On the CC view the calcifications look powdery and spread out, on the MLO view they may layer. I suspect they will show you a ML view because they should layer into a more linear appearance, with a curved bottom **"tea-cupped."** For the purpose of gamesmanship if they show you a ML view on a calcs question - look hard for anything that resembles tea-cupping.
- (2) It's fluid-fluid in a lobule - **due to fibrocystic change.**



Milk of Calcium - Tea Cups on ML

No Calcifications on the Biopsy?

This is a common trick. Apparently **Milk of Calcium needs to be viewed with polarized light to assess birefringence**. Otherwise, you can't see it. I imagine there are several ways to get at that via multiple choice.

Round: The idea is that these things develop in lobules, are usually scattered, bilateral, and benign. When benign (which is most of the time) they *are going to be due to fibrocystic change* (most of the time). The best way I've heard to think about these is the same as a mass. When masses are bilateral, multiple, and similar they are considered benign (BR-2). When a mass is by itself or different it's considered suspicious. Round calcifications are the same way. They are usually bilateral and symmetric (and benign). If they are clustered together by themselves, or new they may need worked up (just like a mass). Remember that if clustered round calcs are on the first mammogram you can BR-3 them.

Suspicious

Amorphous - These things look like powdered sugar, and you should not be able to count each individual calcification. Distribution is key with amorphous calcs (like many other types before). If the calcs are scattered and bilateral they are probably benign, if they are segmental they are probably concerning.

DDx Amorphous Ca⁺²

Fibrocystic Change (*most likely*)
Sclerosing Adenosis
Columnar Cell Change
DCIS (low grade)

Coarse Heterogeneous - These calcifications are countable, but their tips are dull. If you picked one up it would not be poke you. They are usually **bigger than 0.5mm**. Distribution and comparison to priors is always important. They can be associated with a mass (fibroadenoma, or papilloma).

DDx Coarse Heterogeneous Ca⁺²

Fibroadenoma
Papilloma
Fibrocystic Change
DCIS (*low - intermediate grade*)

Fine Pleomorphic - These calcifications are countable, and their tips appear sharp. If you picked one up it would poke you. They are usually **smaller than 0.5mm**. *This morphology has the highest suspicion for malignancy (that would make a good multiple choice question huh?).*

DDx Fine Pleomorphic Ca⁺²

Fibroadenoma (*less likely*)
Papilloma (*less likely*)
Fibrocystic Change
DCIS (*high grade*)

Fine Linear /Fine Linear Branching - This is a distribution that makes fine pleomorphic calcifications even more suspicious. The **DDx narrows to basically DCIS** or an atypical look for secretory calcs or vascular calcs.

Calcifications Associated with Focal Asymmetry/Mass: When you see increased tissue density around suspicious calcifications the chance of an actual cancer goes up. This is sometimes called a **“puff of smoke” sign**, or a “warning shot.” This is a situation where ultrasound is useful, for extent of disease.

Calcifications in/near a Lumpectomy Scar: The local recurrence rate is around 6%. New calcifications with a suspicious morphology (not fat necrosis) deserves a biopsy.

Benign Breast Disease

Mondor Disease: This is a thrombosed vein that presents as a tender palpable cord. It looks exactly like you’d expect it to with ultrasound. You don’t anticoagulate for it (it’s not a DVT). Treatment is just NSAIDS and warm compresses.

Fat Containing Lesions: There are five classic fat containing lesions, all of which are benign. The oil cyst / fat necrosis, hamartoma, galactocele, lymph nodes, and lipoma. Of these 5, only oil cyst/fat necrosis and lipoma are considered “pure fat containing” masses.

- * **Hamartoma** - The buzzword is “breast within a breast.” They have an Aunt Minnie appearance on mammography, although they are difficult to see on ultrasound (they blend into the background).
- * **Gaiactoccele** - Seen in young lactating women. This is typically seen on cessation of lactation. The location is typically sub-areolar. The appearance is variable, but can have an Aunt Minnie look with a fat-fluid level. It’s possible to breast abscess these things up.
- * **Oil Cyst / Fat Necrosis** - These are areas of fat necrosis walled off by fibrous tissue. You see this (1) randomly, (2) post trauma, (3) post surgery. The peripheral calcification pattern is typically “egg shells.” *If you see a ton of them you might think about steatocystoma multiplex* (some zebra with hamartomas).
- * **Lipoma** - These are typically radiolucent with no calcifications. Enlargement of a lipoma is criteria for a biopsy. *
- * **Intramammary' Lymph node:** These are normal and typically located in the tissue along the pectoral muscle, often close to blood vessels. They are NOT seen in the fibroglandular tissue.



Hamartoma /Breast Within a Breast/

Practice Point: *Does she need an ultrasound if it's palpable?* Usually a palpable finding is going to get an ultrasound. If you are under 30 most people will skip the mammo and go straight to ultrasound. One of the exceptions is a fat containing lesion definite benign BR-2er on diagnostic mammography.

Pseudoangiomatous Stromal Hyperplasia (PASH): This is a benign myofibroblastic hyperplastic process (hopefully that clears things up). It's usually big (4-6cm), solid, oval shaped, with well defined borders. Age range is wide and they can be seen between 18-50 years old. Follow up in 12 months (annual) is the typical recommendation.

Pseudoangiomatous Stromal Hyperplasia = Benign thing with a scary sounding name

Fibroadenoma - This is the most common palpable mass in young women. The typical appearance is an oval, circumscribed mass with homogeneous hypoechoic echotexture, and a central hyperechoic band. If it's shown in an older patient it's more likely to have coarse "popcorn" calcifications - which is a buzzword. On MRI, it's T2 bright with a type 1 enhancement (progressive enhancement).

Phyllodes: Although I lumped this in benign disease, this thing has a malignant degeneration risk of about 5-25%. This is a fast growing breast mass. It occurs in an older age group than the fibroadenoma (40s-50s). Biopsy of the sentinel node is not needed, because mets via the lymphatics are so incredibly rare.

Distinguishing Features of Phyllodes Tumor

- * *Rapid Growth*
- * *Hematogenous Mets*
- * *Middle-Age to Older Women*
- * *Mimics a Fibroadenoma*

Cancer

IDC - Invasive Ductal Carcinoma is by far the most common invasive breast cancer, making up about 80-85% of the cases. This cancer is ductal in origin (duh), but unlike DCIS is not confined to the duct. Instead it “invades” through the duct and if not found by the heroic action of mammographers it will progress to distal mets and certain death. Clinically, the most common story is a hard, non-mobile, painless mass. On imaging, the most common look is an irregular, high density mass, with indistinct or spiculated margins, associated pleomorphic calcifications, and an anti-parallel shadowing mass with an echogenic halo on ultrasound.

Invasive Ductal NOS - By far the most common type of breast cancer is the one that is undifferentiated and has no distinguishing histological features. “Not Otherwise Specified” or NOS they call it. These guys make up about 65% of invasive breast cancer.

IDC Subtypes

IDC Types - (Other than NOS)		
Tubular	Small spiculated slow growing mass, with a favorable prognosis .	Often conspicuous on ultrasound. Associated with a Radial Scar . Contralateral breast will have cancer 10-15% of the time.
Mucinous	Round (or lobulated) and circumscribed mass	Uncommon . Better outcomes than IDC-NOS
Medullary	Round or Oval circumscribed mass, without calcifications.	Axillary nodes can be large even in the absence of mets . Typically younger patient (40s-50s). Better outcome than IDC-NOS.
Papillary	Complex cystic and solid .	Axillary nodes are NOT common . Typically seen in elderly people, favors people who are not white, and is the 2 nd most common (behind IDC-NOS).

Multifocal Breast Cancer	Multicentric Breast Cancer
Multiple primaries in the same quadrant (classically same duct system)	Multiple primaries in different quadrants

Synchronous Bilateral Breast Cancer - This is seen in 2-3% of women on mammography, with another 3-6% found with MRI. The risk of bilateral disease is increased in infiltrating lobular types, and multi-centric disease.

DCIS - This is the “earliest form of breast cancer.” In this situation the “cancer” is confined to the duct. Histologists grade it as low, intermediate, or high. Histologists also use the terms “comedo”, and “non-comedo” to subdivide the disease. If anyone would ask, the **comedo type is more aggressive** than the non-comedo types.

Testable Trivia:

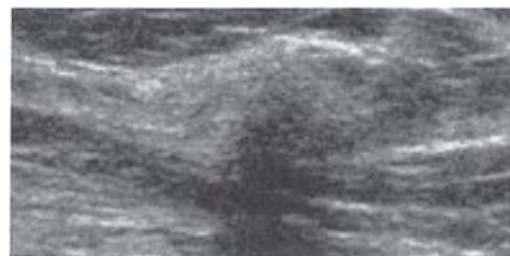
- 10% of DCIS on imaging may have an invasive component at the time biopsy is done
- 25% of DCIS on core biopsy may have an invasive component on surgical excision.
- 8% of DCIS will present as a mass without calcifications
- Most common ultrasound appearance = microlobulated mildly hypoechoic mass with ductal extension, and normal acoustic transmission

If a test writer wants you to come down on this they will show it in 1 of 3 classic ways: (1) suspicious calcifications (fine linear branching or fine pleomorphic - as discussed above), (2) non mass like enhancement on MRI, or (3) multiple intraductal masses on galactography.

Lobular (ILC) : This is the second most common type of breast cancer (IDC-NOS being the most common). It makes up about 5-10% of the breast CA cases. This pathophysiology lends itself well to multiple choice questions.

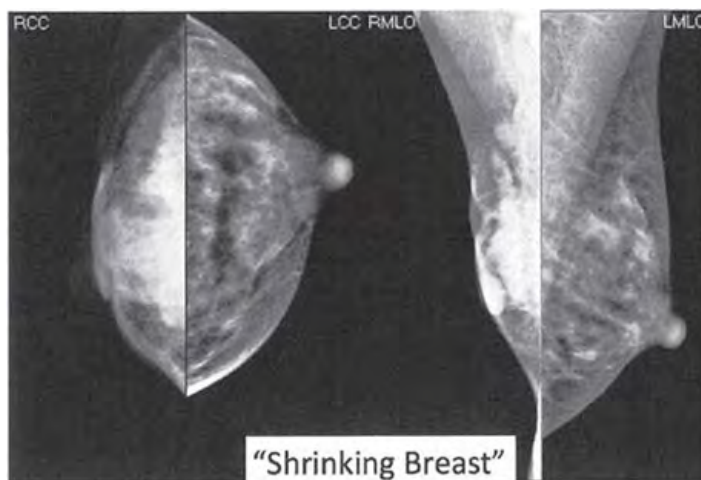
Cell decides to be cancer -> Cells lose “e-cadherin ” -> Cells no longer stick to one another and begin to infiltrate the breast “like the web of a spider” -> This infiltrative pattern does not cause a desmoplastic reaction so it gets missed on multiple mammograms -> Finally someone (you) notices some architectural distortion without a central mass, on the CC view only. You get fancy and call it a “dark scar. ”

On Ultrasound: The typical look is an ill-defined area of shadowing without a mass.

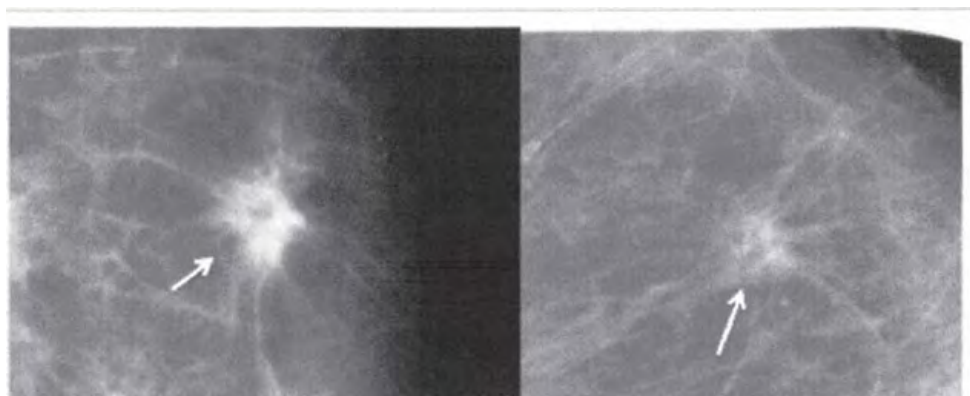


ILC - Shadowing without discrete mass

“Shrinking Breast” - This is a buzzword for ILC. The breast isn’t actually smaller, it just doesn’t compress as much. So when you compare it to a nonnal breast, it appears to be getting smaller. On physical exam, this breast may actually look the same size as the other one.



Dark Star



"Dark Star" - Distortion without a central mass

Architectural distortion without a central mass.

The DDx includes: lobular carcinoma, radial scar, surgical scar, and IDC-NOS.

ILC vs *IDC*: *ILC* is more often multifocal. *ILC* less often mets to the axilla. Instead, it likes to go to strange places like peritoneal surfaces. *ILC* more often has positive margins, and more often treated with mastectomy although the prognosis is similar to *IDC*.

Things to know about ILC:

- * It presents later than IDC
- * Tends to occur in an older population
- * It often is only seen in one view (the CC - as it compresses better)
- * Calcifications are less common than with ductal cancers
- * Mammo Buzzword = Dark Scar
- * Mammo Buzzword = Shrinking Breast
- * Ultrasound Buzzword = Shadowing without mass
- * On MRI - washout is less common than with IDC
- * Axillary mets are less common
- * Prognosis of IDC and ILC is similar (*unless it's a pleomorphic ILC, - which is very aggressive*)
- * More often multifocal (compared to IDC)

Inflammatory Breast Cancer: The prognosis is usually terrible. They will try and do chemotherapy prior to surgery because the chance of a positive margin is so high. The mastectomy is done for “local control”, which just sounds awful. A swollen red breast is what you are going to see, and as I’ll discuss below under the “symptomatic breast” section your differential is mastitis vs this. “Skin thickening” is a mammography buzzword (non-specific). The inflammation associated with inflammatory breast cancer can actually improve with antibiotics, but does NOT resolve. So, don’t be fooled (in the real world or on a multiple choice test). A dermal biopsy is sometimes needed if you can’t find an underlying mass.

Pagets - Paget’s disease of the breast is a high yield topic. It is basically a carcinoma in situ of the nipple epidermis. About 50% of the time the patient will have a palpable finding associated with the skin changes.

Things to know about Breast Pagets:

- * Associated with high grade DCIS
- * Wedge biopsy should be done on any skin lesion that affect the nipple-areolar complex that doesn’t resolve with topical therapy.
- * Pagets is NOT considered T4. The skin involvement does not up the stage in this setting.

High Risk Lesions:

There are 5 classic high risk lesions that must come out after a biopsy; Radial Scar, Atypical Ductal Hyperplasia, Atypical Lobular Hyperplasia, LCIS, and Papilloma.

Radial Scar: This is not actually a scar, but does look like one on histology. Instead you have a bunch of dense fibrosis around the ducts giving the appearance of architectural distortion (dark scar).

Things to know:

- * *This is high risk and has to come out*
- * *It's associated with DCIS and/or IDC 10%-30%*
- * *It's associated with Tubular Carcinoma**

Atypical Ductal Hyperplasia (ADH): This is basically DCIS but lacks the quantitative definition by histology (< 2 ducts involved). It comes out (a) because it's high risk and (b) because DCIS burden is often underestimated when this is present. In other words, about 30% of the time the surgical path will get upgraded to DCIS.

Lobular Carcinoma in situ (LCIS): This is classically occult on mammogram. "An incidental finding" is sometimes a buzzword. The best way to think about LCIS is that it can be a precursor to ILC, but isn't obligated to be. The risk of conversion to an invasive cancer is less when comparing DCIS to IDC. Just like pleomorphic ILC is worse than regular ILC, a pleomorphic LCIS is mo' badder than regular LCIS.

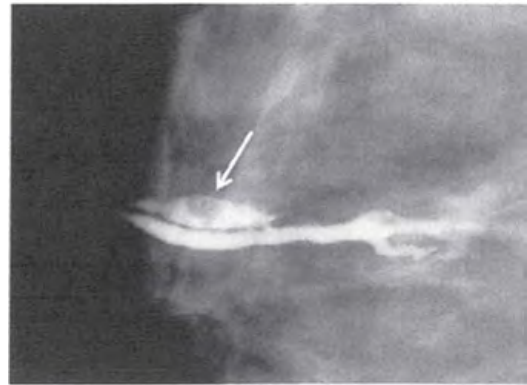
Atypical Lobular Hyperplasia (ALH): This is very similar to LCIS, but histologists separate the two based on if the lobule is distended or not (no with ALH, yes with LCIS). It's considered milder than LCIS (risk of subsequent breast CA is 4-6x higher with ALH, and 1 lx higher with LCIS). For the CORE, the answer is excision. In the real world, some people do not cut these out, and it's controversial.

Papilloma: A few most commons come to mind with this one. Most common intraductal mass lesion. Most common cause of blood discharge. You typically see these in women in their late reproductive years / early menopausal years (average around 50). The classic location is the subareolar region (1cm from the nipple in 90% of cases).

On Mammo: Often normal, can have calcifications.

On US: Well-defined smooth walled hypo-echoic mass. Maybe cystic with solid components. Also, tends to have associated duct dilation.

On Galactography: Solitary filling detected, with dilated duct.

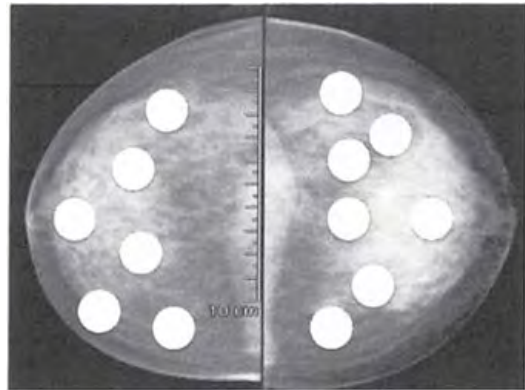


Papilloma — *Solitary filling defect with dilated duct*

Multiple Papillomas: These tend to be more peripherally. On mammography it's gonna be a mass(es) or cluster of calcifications without a mass.

Multiple Masses:

To call multiple masses you need to have multiple (at least 3) bilateral well circumscribed masses without suspicious features. **This gives you a BR-2.** One common trick is to show multiple unilateral masses, that doesn't fly - they have to be bilateral.



Symptomatic Breasts

Breast Pain: This is super common and typically cyclic (worse during the luteal phase of the menstrual cycle). Pain in both breasts that is cyclical does not need evaluation, it needs a family medicine referral for some “therapeutic communication.” Focal non-cyclic breast pain may warrant an evaluation.

Symptoms that can be worrisome for cancer include: skin dimpling, focal skin thickening, and nipple retraction.

Non-Focal Skin Thickening / Breast Edema: This is usually the result of benign conditions (congestive heart failure, renal failure). For multiple choice tests it will always be bilateral (in the real world you can sleep on one side and have asymmetric edema). As long as the breast isn't red, you can feel confident that it will be benign. On mammography you will see trabecular thickening (diffuse, and favoring the dependent portions of the breast).

Breast Inflammation: The swollen red breast. This finding has a differential of two things: (1) mastitis / abscess, (2) inflammatory breast cancer.

- * **Mastitis / Abscess:** This is a swollen red breast which is painful (Inflammatory breast CA is often painless). Patients are usually sick as a dog. Obviously it's associated with breast feeding, and is more common in smokers and diabetics. Abscess can develop (usually Staph A.).
- * **Inflammatory Breast Cancer:** As discussed above, this has a terrible prognosis. The general rule is that a breast that doesn't respond to antibiotics gets a skin biopsy to exclude this. The typical age is 40s-50s. You are going to have an enlarged, red, breast with a “peau d'orange” appearance. The breast is often NOT painful, despite its appearance. Mammogram might show a mass (or masses), but the big finding is diffuse skin and trabecular thickening. The treatment is fair game for multiple choice because it is different than normal breast cancer. Instead of going to surgery first, inflammatory breast cancer gets “cooled down” with chemo and or radiation - then surgery.

Discharge: Women present with nipple discharge all the time, it's usually benign (90%). **The highest yield information on the subject is that; spontaneous, bloody, discharge from a single duct is your most suspicious feature combo. Serous discharge is also suspicious.** The risk of discharge being cancer is directly related to age (very uncommon under 40, and more common over 60).

BAD Discharge = Spontaneous, Bloody, from a Single Duct

Milky Discharge: Milky discharge is NOT suspicious for breast cancer but can be secondary to thyroid issues or a pituitary adenoma (prolactinoma). Any medication that messes with dopamine can stimulate prolactin production - (antidepressants, neuroleptics, reglan)

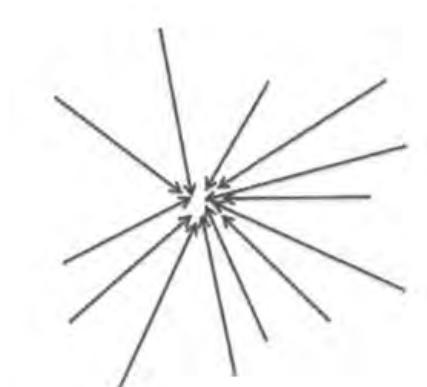
Causes of Discharge (Not Milky)	
Benign Causes	Worrisome Causes
Pre-Menopausal Woman = Fibrocystic Change	Intraductal Papilloma (90%) - single intraductal mass near nipple
Post Menopausal Women = Ductal Ectasia	DCIS (10%) - think about multiple intraductal masses

Ductal Ectasia - The most common benign cause of nipple discharge in a post menopausal women. On galactography you will see dilated ducts near the subareolar region, with progressive attenuation more posterior.

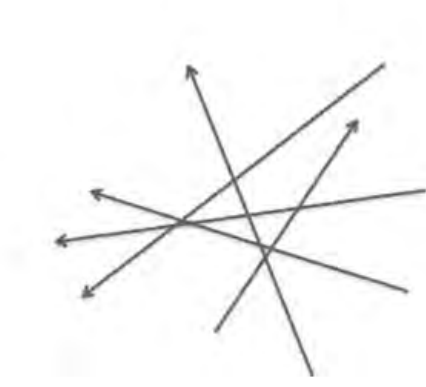
Architectural Distortion (AD)

AD: We are talking about distortion of the normal architecture without a visible mass. This manifests in a few ways, including focal retraction, distortion of the edge of the parenchyma, or *radiation of the normal thin lines into a focal point*.

Architectural Distortion vs Summation Artifact: This is the primary differential consideration, with summation of normal vessels, ducts, and ligaments being much more common. The difference is summation should NOT radiate to a central point (AD will).



AD - All lines radiate to a point



Summation - Lines continue past each other

Surgical Scar vs Something Bad: Scars should progressively get lighter and harder to see. Some people say that in 5-10 years a benign surgical scar is often difficult to see. Lumpectomy scars tend to stick around longer than a benign biopsy. Basically, look at the priors if it is a surgical scar it better be getting less dense. If it's increasing you gotta stick a needle in it.

Work Up of AD: If you see it on a screener you will want to BR-0 it, and bring it back for spot compression views. If it persists just know you are either going to BR-4 or BR-5 it (unless you know it's a surgical scar). You should still ultrasound it for further characterization (may help you decide between a 4 and a 5).

Ultrasound Trivia: The use of harmonic tissue imaging can make it easier to see some lesions. Be aware that compound imaging can make you lose your posterior features, especially when they are soft to start with - like the shadowing of an ILC. Remember, even if you see nothing this gets a biopsy. Harmonics can also make not so simple cysts look simple.

Things to Know for AD:

- * Radiating lines to a single point = AD
- * AD + Calcifications = IDC + DCIS
- * AD without Calcifications = ILC
- * Even with no ultrasound or MRI correlate, AD gets a biopsy.
- * Never ever ever ever ever BR-3 an area of AD.
- * Even if it has been there a while, it still needs worked up.
- * Remember ILC can grow slow.
- * Surgical scars should get less dense with time... not more dense.

Lymph Nodes

You found a breast cancer - now what? Before you make the patient cry, it's time to stage the disease. Ultrasound her arm pit. About 1 in 3 times you are going to find abnormal nodes.

Unilateral vs Bilateral: This can help you if you are thinking this could be systemic. Unilateral adenopathy should make you worry about a cancer (especially if they have a cancer on that side).

Should I Biopsy It? Some people will recommend biopsy if you have the following abnormal features.

- * Cortical Thickness greater than 2.3mm (some people say 3mm)
- * Loss of Central Fatty Hilum - “most specific sign”
- * Irregular Outer Margins.

Staging Trivia: Level 1 and Level 2 nodes are treated the same. Rotter nodes are treated as Level 2. Level 3 and supraclavicular nodes are treated the same.

Special “Sneaky” Situations:

Gold Therapy: Back in the stone ages they treated rheumatoid arthritis with “chrysotherapy.” What they can do is show you an “Aunt Minnie” type picture with very dense calcifications within the node.

Snow Storm Nodes: Another Aunt Minnie look is the silicone infiltration of a node from either silicone leaking or rupture.



Gold
-High Density Calcifications



Silicone Nodes
-Snow Storm Appearance

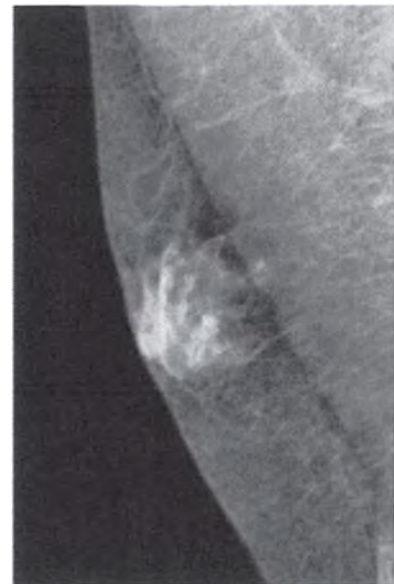
The Male Breast

There is no more humiliating way to die for a man than breast cancer. The good news is male breast cancer is uncommon, the bad news is that when it occurs it is often advanced and invasive at the time of diagnosis.

The male breast does NOT have the elongated and branching ducts, or proliferated lobules that women have. This is key because **men do NOT get lobule associated pathology; lobular carcinoma, fibroadenoma, or cysts.**

Gynecomastia: This is a non-neoplastic enlargement of the epithelial and stromal elements in a man's breast. It occurs "physiologically" in adolescents, affecting about 50% of adolescent boys, and men over 65. If you aren't 13 or 65 it's considered embarrassing and you should hit the gym. If you're between those ages it's considered pathology and associated with a variety of conditions (spironolactone, psych meds, marijuana, alcoholic cirrhosis, testicular cancer). There are three patterns (nodular is the most common). Just think **flame shaped, behind nipple, bilateral but asymmetric, and can be painful**. Things that make you worry that it's not gynecomastia include not being behind the nipple, eccentric location, and calcification.

Patterns of Gynecomastia	
Nodular (most common)	"Flame Shaped" centered behind the nipple, radiating posterior as it blends into the fat. Breast is often tender. Usually lasts less than 1 year.
Dendritic	Resembles a branching tree. This is a chronic fibrotic pattern. Usually not tender.
Diffuse Glandular	Mammographic pattern looks like a woman's breast (diffuse increase in density). You see this in men receiving estrogen treatment.



Gynecomastia

Pseudogynecomastia “*Bitch Tits*” - This is an increase in the fat tissue of the breast (not glandular tissue). There will NOT be a discrete palpable finding, and the mound of tissue will not be concentric to the nipple.

Lipoma - After gynecomastia a lipoma is the second most common palpable mass in a man.

Male Breast Cancer: It's uncommon in men, and very uncommon in younger men (average age is around 70). About 1 in 4 males with breast cancer have a BRCA mutation (BRCA 2 is the more common). Other risk factors include Klinefelter Syndrome, Cirrhosis, and chronic alcoholism. The classic description is eccentric but near the nipple. It's almost always an IDC-NOS type. DCIS can occur but is very rare in isolation. On mammography it looks like a breast cancer, if it was a woman's mammogram you BR-5 it. On ultrasound it's the same thing, it looks like a BR-5. Having said that nodular gynecomastia can look suspicious on ultrasound.

Things that make you think it's breast cancer:

- * *Eccentric to Nipple*
- * *Unilateral*
- * *Abnormal Lymph nodes*
- * *Calcifications*
- * *Looks like breast cancer*



Male Breast CA

Some trivia on calcifications: Micro-calcifications alone are uncommon in men. When you see them they are less numerous, coarser, and associated with a mass (25% of male breast cancers have calcifications).

Should men get screening mammograms? Honestly, women shouldn't even get them (according to the New England Journal of Medicine). This remains controversial, with the bottom line being this; only Klinefelter patients approach the screening range with regards to risk. As a point of testable trivia: **males with gynecomastia from gender reassignment on hormone therapy are not high enough risk for screening mammograms.** Obviously, if they have a palpable they can get a diagnostic work up.

Implants

Basic Overview: There are two types, saline and silicone. They both can rupture, but no one really gives a shit if saline ruptures. Saline does not form a capsule, so you can't have intracapsular rupture with saline. There is no additional imaging past mammo for saline rupture, and you just follow up with primary care / plastic surgeon. You can tell it's saline because you can see through it. For silicone you can have both intra and extra capsular rupture. You can only see extra on a mammogram (can't see intra). So, extra creates a dense "snow storm" appearance on US. Intra creates a "step ladder" appearance on US and a "linguine sign" on MRI. MRI is done with FS T2 to look at implants.

Big Points:

- * You CAN have isolated intracapsular rupture.
- * You CAN NOT have isolated extra (it's always with intra).
- * If you see silicone in a lymph node you need to recommend MRI to evaluate for intracapsular rupture

Implant Location: There are two subtypes:

- * *Subglandular (retromammary)*: Implant behind breast tissue, anterior to pectoral muscle
- * *Subpectoral (retropectoral)*: Implant between pectoralis major and minor muscles

Silicone Implants

The body will form a shell around the foreign body (implant), which allows for both intracapsular and extracapsular rupture (an important distinction from saline). About 25% of the time you will see calcifications around the fibrous capsule.

Things to know:

- * Implants are NOT a contraindication for a core needle biopsy
- * Implants do NOT increase the risk for cancer.

Saline Implants

There are also subglandular and subpectoral subtypes. You can tell the implant is saline because you can see through it. Implant folds and valves can also be seen. If it ruptures no one really cares (other than the cosmetic look). The saline is absorbed by the body, and you have a collapsed implant. A practical point of caution, be careful when performing a biopsy in these patients - even a 25g FNA needle can burst a saline implant.

Implant Complications:

Generally speaking MRI is the most accurate modality for evaluating an implant.

Capsular Contracture: This is the **most common complication of implants**. It occurs secondary to contraction of the fibrous capsule, and can result in a terrible cosmetic deformity. You see it in both silicone and saline implants, but is **most common in subglandular silicone implants**. On mammo it looks like rounding or distortion of the implant (comparisons will show progression).

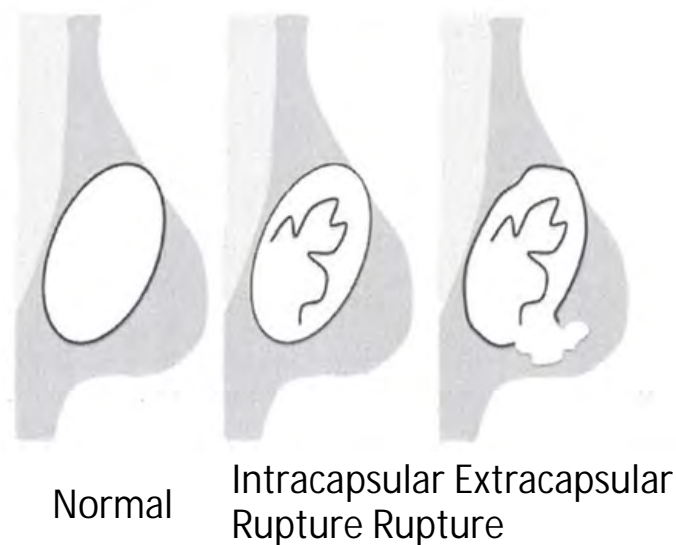
Gel Bleed: Silicone molecules can (and do) pass through the semi-permeable implant shell coating the exterior of the surface. This does NOT mean the implant is ruptured. The classic look is to show you silicone in the axillary lymph nodes (*remember I showed a case of this under the lymph node section*). Even with axillary lymph nodes, this does NOT mean it has ruptured.

Rupture: As a point of testable trivia the number one risk factor for rupture is age of the implant. Rupture does not have to be post traumatic, it can occur spontaneously. Rupture with compression mammography is actually rare.

- **Saline:** Saline rupture is usually very obvious (deflated boob). It doesn't matter all that much (except cosmetically), as the saline is just absorbed. On mammo, you will see the "wadded up" plastic wrapper. *They could easily write a question asking you what modality you need to see a saline rupture. The answer would be plain mammo (you don't need ultrasound or MRI).*
- * **Silicone:** This is a more complicated matter. You have two subtypes; isolated intracapsular and intracapsular with extracapsular.

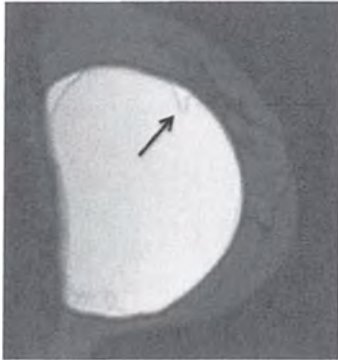
○ *Isolated Intracapsular:* This will be occult on physical exam, mammography and possibly ultrasound. You might see a stepladder on Ultrasound. MRI is way more sensitive.

Intracapsular with Extracapsular Rupture: This is usually obvious on mammogram with dense silicone seen outside the capsule. The contour of a normal intact implant is smooth. Silicone outside the implant can go to lymph nodes. On ultrasound you want to know the buzzword **“snow storm” pattern - which is really echogenic with no posterior shadowing.** A sneaky trick is to show a lymph node with a snow storm appearance on ultrasound. On MRI extracapsular silicon is T1 dark, and T2 bright. Lastly, a very important concept is that ***you cannot have isolated extracapsular rupture. If it is extracapsular, then it's also intracapsular.***

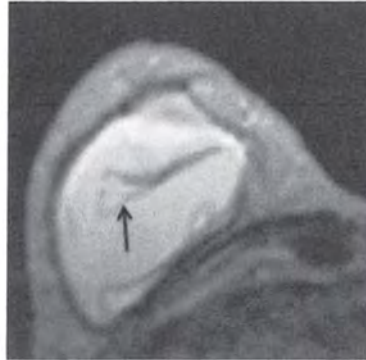


Signs of Intracapsular Rupture	
Stepladder Sign	Seen on ultrasound. Multiple parallel echogenic lines within the sonolucent silicone.
Linguine Sign	Seen on MRJ. Curvy low signal line within the implant (that looks like linguine). It's basically the shell floating in the silicone (with the fibrous shell holding the silicone together).
Keyhole Sign / Noose Sign / Inverted Teardrop Sign	Seen on MRI. Silicone on both sides of a radial fold.
Salad Oil Sign	Seen on MRI. Caused by rupture of the inner lumen of a double-lumen implant (causes saline and silicone to rupture).
Subcapsular Line Sign	Seen on MRI. Silicone adjacent to both surfaces of the ruptured shell.

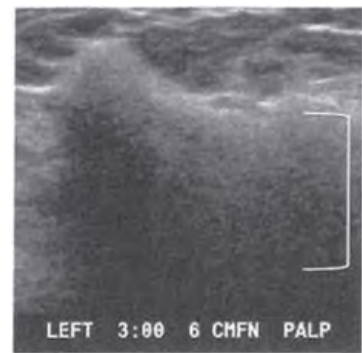
Classic Implant Malfunction Pictures:



Noose/Inverted Tear Drop Sign
- Associated with Gel Bleed



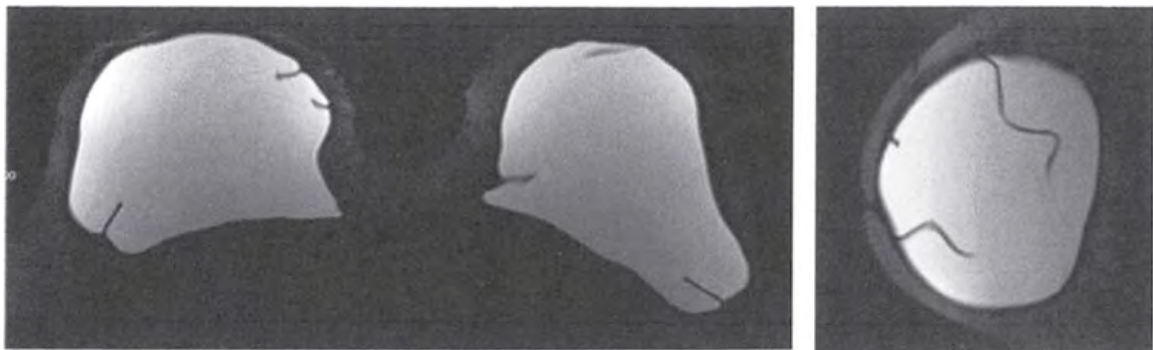
Linguine Sign
- Intracapsular Rupture



Snowstorm Sign
- Extracapsular Silicone

Radial Folds - The Mimic of Rupture:

Radial folds are the normal infoldings of the elastomer shell. They are the primary mimic for the linguine sign of intracapsular rupture. To tell them apart ask yourself “*do the folds connect with the periphery of the implant?*” Radial folds should always do this (linguine does not).



Radial Folds - *All lines connect to the periphery of the implant*

Screening Mammograms in women with Implants:

You get 4 views of each breast (CC, MLO, implant displaced CC, implant displaced MLO). Obviously sensitivity is decreased in women with implants. Implants are easier to displace if they are subpectorial (so they have better sensitivity than subglandular).

The Post Operative Breast

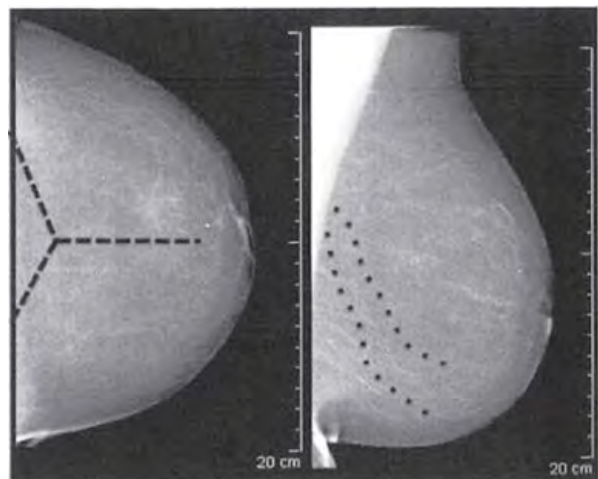
Reduction Mammoplasty and Mastopexy

Reduction Mammoplasty - Yes, there is actually a subpopulation of women who want SMALLER breasts. I know, it sounds impossible to believe (from a man's perspective). Mammoplasty is done to reduce breast size.

Mastopexy - This is a "breast lift." This is **just a removal of skin**. Women get this done to address floppy "ptotic" boobs.

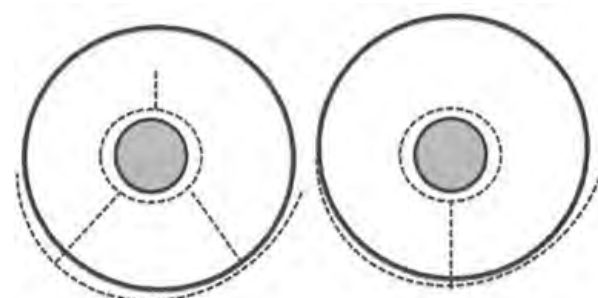
Normal Findings Post Mastopexy:

- *Swirled Appearance Affecting Inferior Breast*
- *Fat Necrosis / Oil Cysts*
- *Isolated Islands of Breast Tissue*



Typical Changes from Mammoplasty

Keyhole Incision - This is done for both mammoplasty and mastopexy, creating a "swirled" appearance in the inferior aspect of the MLO.



Mammoplasty Keyhole Incision

Mastopexy Keyhole Incision

Surgical Biopsy / Radiation

Terminology:

- **Lumpectomy** - Surgical Removal of Cancer (palpable or not)
- **Excisional Biopsy** - Surgical Removal of **Entire** Lesions
- **Incisional Biopsy** - Surgical Biopsy of a **Portion** of the Lesion

Post Biopsy Changes

The first post operative mammogram is usually obtained around 6-12 months after biopsy. The key is that **distortion and scarring are worst on this fdm, and should progressively improve**. On ultrasound, scars are supposed to be thin and linear. If they show you a focal mass like thickening in the scar - you've gotta call that suspicious for local recurrence.

Fat necrosis and benign dystrophic calcifications may evolve over the first year or two, and are the major mimics of recurrence. Fat necrosis can be shown on MR (T1 / T2 bright, and then fat sat drops it out).

Risk of Recurrence / Residual Disease:

Numerical Trivia: **Local recurrence occurs 6-8%** of the time, when women have breast conserving therapy. The **peak time for recurrence is 4 years** (most occur between **1-7**). **Without radiation local recurrence is closer to 35%.**

Residual Calcs: Residual calcifications are not good. Supposedly residual calcifications near or in the lumpectomy bed correlates with a local recurrence rate of 60%.

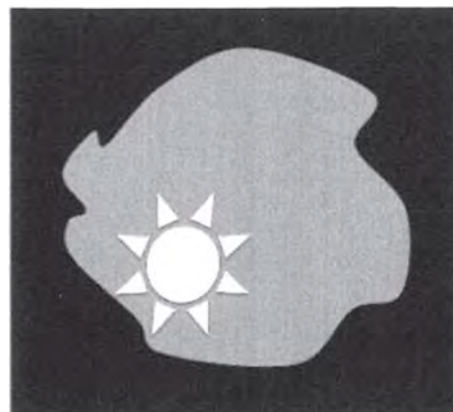
New Calcs: When it does reoccur, something like 75% of DCIS will come back as calcifications (no surprise). The testable pearl is the **benign calcifications tend to occur early (around 2 years), vs the cancer ones which come back around 4 years.**

Sentinel Node Failure: Sentinel node biopsy works about 95% of the time (doesn't work 5% of the time). So about 5 times in 100 you are going to have a negative SNLB that presents later with an abnormal armpit node.

Tissue Flap: The cancer is not going to start in the belly fat / muscle. The cancer is going to come from either the residual breast tissue or along the skin scar line. Screening of the flaps is controversial - with some saying it's not necessary. The need for screening of tissue flaps is not going to be asked. If you get asked anything it's "where the recurrent is coming from / going to be?"

Specimen Radiography

If the path report says “close margins” or “positive margins” there is a very high chance you are going to have cancer still in the breast. If you are shown a specimen radiograph there are two things you need to look at in real life and on multiple choice: (1) is the mass / calcifications on the sample and (2) is the mass / calcifications near the edge or touching the edge. *If the mass is at the edge, the chance of incomplete excision is going to be near 80%. The “next step ” would be to call the surgeon in the OR and tell him/her that.*



*Specimen Radiograph . Cancer at the Margin
High chance of positive margin*

Post Radiation Changes:

Practical Point (*the before picture*): The pre-radiation mammogram is very important. If you can identify residual disease on it, the patient has many more treatment options. If you discover the residual disease after the radiation therapy has been given, you’ve forced the patient to undergo mastectomy.

Radiation Changes: You are going to see skin thickening and trabecular thickening. This is normal post radiation, and should peak on the first post-RT mammogram.

This would be a classic testable scenario:

- Film 1 Post RT: You see skin thickening / trabecular thickening
- Film 2: Skin thickening / trabecular thickening is better
- Film 3: Skin thickening / trabecular thickening is worse * - this is recurrent disease (maybe inflammatory breast CA).

Staging/ Surgical Planning

Breast Cancer Staging: The staging is based on size from T1-T3, then invasion for T4.

- T1 = < 2cm.
- T2 = 2-5cm
- T3 = > 5cm
- T4 = “Any size” with chest wall fixation, skin involvement, or inflammatory breast CA. **Remember that Pagets is NOT T4.*

The contraindications for breast conservation are high yield.

Contraindications for Breast Conservation

Inflammatory Cancer,
Large Cancer Size Relative to Breast,
Multi-centric (multiple quadrants),
Prior Radiation Therapy,
Contraindication to Radiation Therapy (collagen-vascular disease).

Breast MRI

Breast MRI can be used for several reasons; High risk screening, extent of disease (known cancer), axillary mets with unknown primary, diagnostic dilemmas, and possible silicone implant rupture. **The big reason is for screening.**

Who gets a screening MRI ?

- * *People with a lifetime risk greater than 20-25%*
- * *Includes people who got 20Gy of radiation to the chest as a child*

How do you estimate this risk, to decide who is 20-25%?

- * *You use one of the risk models that includes family history (NOT the Gail model). If the question is which of the following is Not one to use ? The answer is Gail. If the question is which of the following do you pick? I'd chose Tyrer-Cuzick, it's probably the best one out now.*

Parenchyma Enhancement:

- * *Is it normal ? - Yes*
- * *Where is it most common ? - Posterior Breast in the upper outer quadrant*
- * *How do you reduce it? - Do the MRI during the first part of the menstrual cycle (day 7-20).*
- * *What does Tamoxifen do? - Tamoxifen will decrease background parenchyma uptake. **Then it causes a rebound.***

Foci:

- * *How is it defined? Round or oval, circumscribed, and **less than 5mm.***
- * *Are they high risk? Usually not. Usually they are benign (2-3%).*
- * *What would make you biopsy one? Seemed different than the rest, ill-defined borders, or **suspicious enhancement.***

Masses:

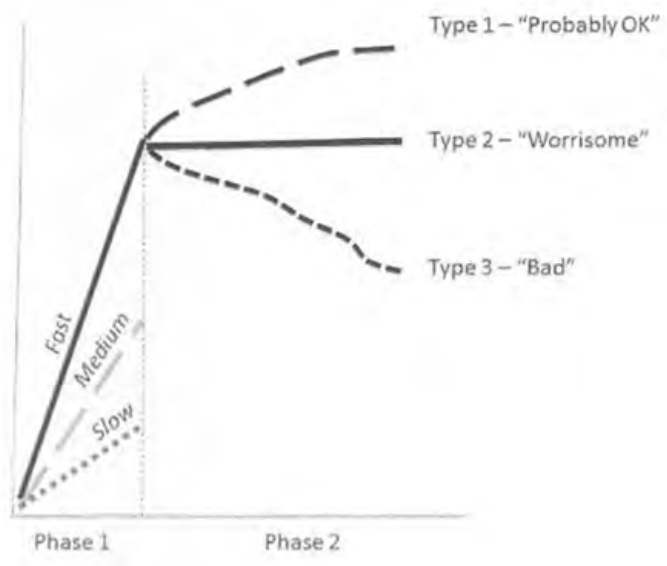
- * These are defined as being 5mm or larger. They have definable vocabulary for their features (round, oval, indistinct, etc...)
- * *When are these bad?* They are bad when you call them bad words. Irregular shape, speculated margins, heterogeneous or rim enhancement. Once you say those words you are going to have to biopsy them, because **morphology trumps kinetics**. It doesn't matter what the kinetics shows, you must biopsy suspicious morphology.
- * *When is kinetics helpful?* When you are on the fence. If you have benign morphology and you have suspicious kinetics - you probably are going to need to biopsy that also.

Kinetics:

- * Breast kinetics are performed in two portions:
 - o (1) Initial upslope phase that occurs over the first 2 minutes. This is graded as slow, medium, or rapid (fast).
 - o (2) The washout portion which is recorded sometime between 2 minutes and 6 minutes (around about). These are graded as either continued rise "type 1", plateau "type 2", or rapid washout "type 3". *

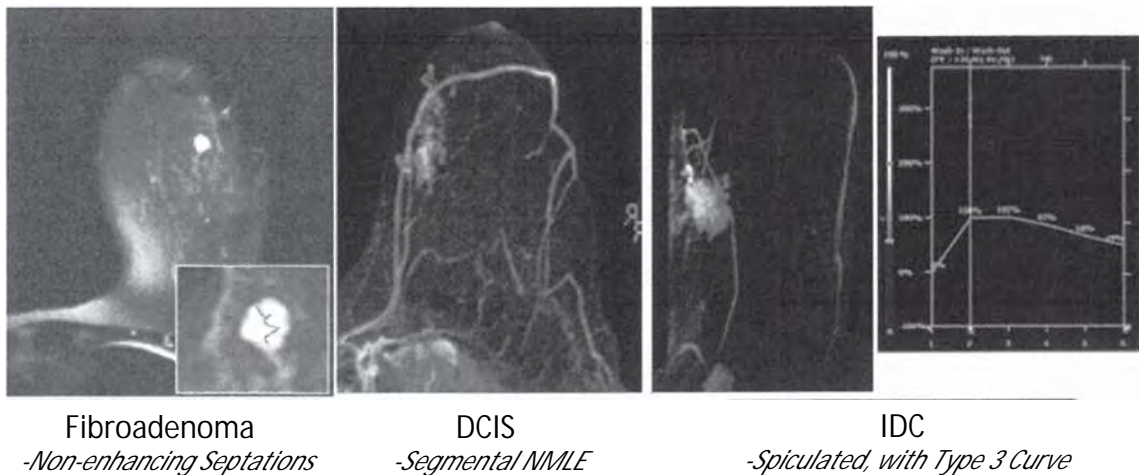
* Risk of Cancer:

- o Type 1 Curve: 6%
- o Type 2 Curve: 7%-28%
- o Type 3: 29% or more.



Classic Looks:

- **Fibroadenoma:** These things are classically T2 bright, round, with “non-enhancing septa”, and a type 1 curve.
- **DCIS:** Clumped, ductal, linear or segmental **non-mass likely enhancement**. Kinetics are typically not helpful for DCIS.
- **IDC:** Spiculated irregular shaped masses, with heterogeneous enhancement and a type 3 curve.
- **ILC:** Doesn't always show enhancement.



T2 Bright Things:

- * Usually T2 Bright = Benign. Things that are T2 Bright include: Cysts, Lymph nodes, fat necrosis, Fibroadenoma.
- * The exception (*anytime I say the word “except ” you need to think high yield!*): **Colloid Cancer**, and Mucinous Cancer can be T2 bright.

Pure Trivia:

- * *If you have a patient with known breast CA, how often do you find a contralateral breast CA?* - Answer is 0.1-2% via mammogram, and 3-5 % by MRI.
- *Never BR-0 an MRI case.* This is as much workup as you are going to get, so just call it benign or biopsy it. You can actually BR-0 something if you really want to prevent a biopsy - possible lymph node - US and mammo to confirm benign sorta situation. This is still kinda weak. For the purpose of multiple choice, think twice before you BR-0 a MRI case.
- # *Spiculated margins = 80% malignancy.* This is the **single most predictive feature of malignancy.**

Implants: - As discussed above.

MQSA / Medial Audit

The U.S. Food and Drug Administration Mammography Quality Standards Act (MQSA) - yes that is a real thing - demands a medical audit and outcome analysis be performed once a year. You are forced to follow up patients with positive mammos, and correlate pathology with biopsy results (so you can see how much benign disease you biopsy and how much fear / anxiety you generate). You have to grade the biopsy with the risk category (you can't accept benign results with a BR-5).

MQSA and Other Crap they could ask:

- 3 months of mammography is required during residency training
- The recall rate should be less than 10%
- Mammography facilities are required to provide patients with written results of their mammograms in language that is easy to understand. Also known as a "lay report."
- A consumer complaint mechanism is required to be established in mammography facilities to provide patients with a process for addressing their concerns.
- Patients can obtain their original mammograms, not copies, when they are needed.
- For cases in which a facility's mammograms are determined to be substandard and a risk to public health, facilities will notify the patients and their doctors and suggest an appropriate plan of action.

- The “Interpreting Physician” is ultimately responsible for the Quality Control program.
- The required resolution of line pairs is 13 lp/mm in the anode to cathode direction and 11 pairs in the left right direction
- To make it pass image quality; must show 4 fibers, 3 microcalcification clusters, and 3 masses, plus “acceptable artifacts”.
- The dose phantom is 50% glandularity, 4.2 cm thick, and is supposed to have a dose less than 3 mGy per image (+ grid).
- Don’t get it twisted; there are no patient dose limits in mammography, only a phantom dose. A dense breast can result in a higher patient dose, which could easily exceed 3 mGy/view.
- Typical patient and phantom doses are about 2 mGy per view, or 4 mGy for a two view screening examination.
- The typical (average) compressed breast is 6 cm, glandularity of 15 to 20%.
- Digital systems generally uses higher beam qualities which results in lower doses;
- Digital mammography does not use fixed dose (screen-film); can use as much (or little) radiation as deemed appropriate.

Specific Tasks that the ABK thinks You Should Memorize	
Processor QC	Daily
Darkroom Cleanliness	Daily
Viewbox Conditions	Weekly
Phantom Evaluation	Weekly
Repeat Analysis	Quarterly
Compression Test	Semi-Annually
Darkroom Fog	Semi-Annually
Screen-Film Contrast	Semi-Annually

Appropriate Target Range for Medical Audit	
Recall Rate	5-7%
Cancers/ 1000 Screened	3-8
PPV for Biopsy Recommendations	15-35%

INDEX

11 Neuroradiology

Prometheus Lionhart, M.D.



Neuroradiology has one of the deepest wells for obscure trivia. A lot of neuro is differential diagnosis, which I've stated over and over again makes for a bad multiple choice question. I think the questions will mainly fall into two categories (1) Anatomy / Aunt Minnie - What is it? and (2) Associated trivia / syndrome

High Yield Topics:

- Anatomy - Especially skull base, angiography, and head and neck
- Syndromes: NF-1, NF-2, VHL, TS
- Associations / Typical Findings; what likes to calcify, what is cystic, etc...
- Congenital Brain - These make for good "what is it?" questions

Section 1: Brain

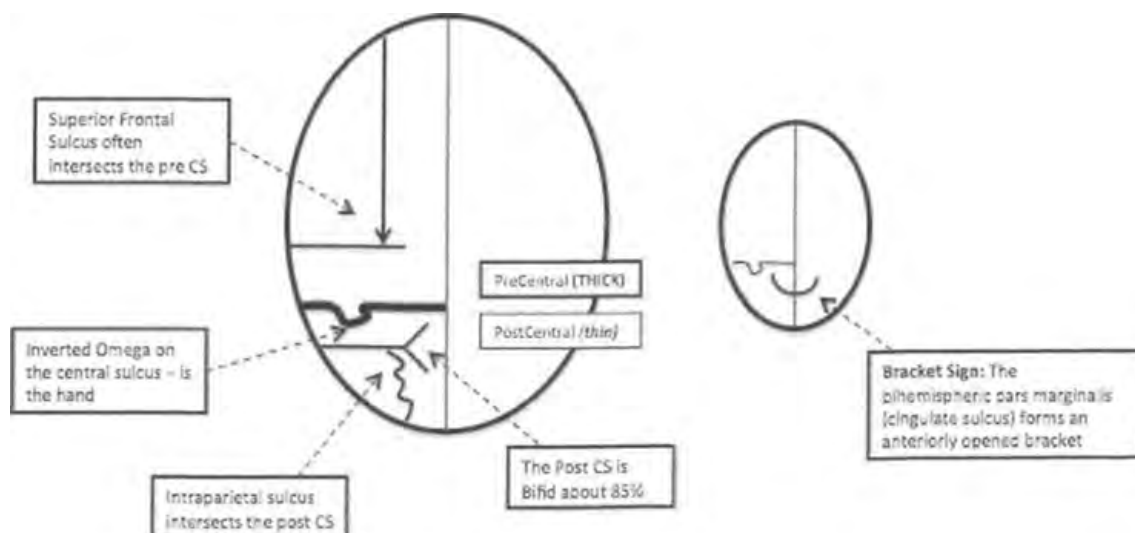
Anatomy

There is a ton of anatomy that can be asked on a multiple choice test. My idea is to break it down into three categories: (1) soft tissue - brain parenchyma (*including normal development*), (2) bony anatomy - which is basically foramina, and (3) vascular anatomy.

Soft Tissue Brain Anatomy:

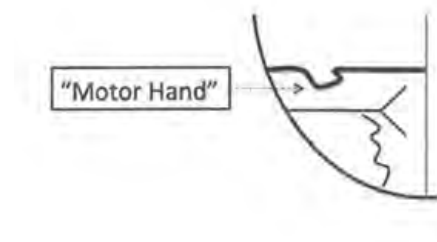
Ways to locate the central sulcus:

- Superior frontal sulcus / Pre-central sulcus sign: The posterior end of the precentral sulcus, joins the pre-central sulcus
- Inverted omega (sigmoid hook) corresponds to the motor hand
- Pars Bracket sign: The bihemispheric symmetric pars marginalis (cingulate sulcus) forms an anterior opened bracket about 95% of the time.
- Bifid posterior central sulcus: Posterior CS has a bifid appearance about 85%
- *Thin post-central gyrus sign* - The precentral gyrus is thicker than the post central gyrus (ratio 1 : 1.5).
- *Intersection* - The intraparietal sulcus intersects the post central sulcus (works almost always)
- *Midline sulcus sign* - The most prominent sulcus that reaches the midline is the central sulcus (works about 70%).



Homoculous Trivia:

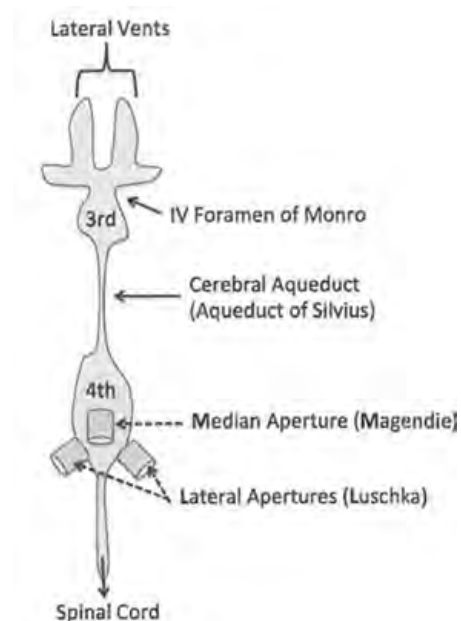
- The inverted omega (posteriorly directed knob) on the central sulcus / gyrus designates the motor cortex controlling hand function.
- ACA territory gets legs, MCA territory hits the rest.



Normal Cerebral Cortex: As a point of trivia, the cortex is normally 6 layers thick, and the hippocampus is normally 3 layers thick. I only mention this because the hippocampus can look slightly brighter on FLAIR compared to other cortical areas, and this is the reason why (supposedly).

Dilated Perivascular Spaces (Virchow-Robins): These are fluid filled spaces that accompany perforating vessels. They are a normal variant and very common. They can be enlarged and associated with multiple pathologies; mucopolysaccharidoses (Hurlers and Hunters) / 'gelatinous pseudocysts' in cryptococcal meningitis, and atrophy with advancing age. They don't contain CSF, but instead have interstitial fluid. The common locations for these are: around the lenticulostriate arteries in the lower third of the basal ganglia, in the centrum semiovale, and in the midbrain.

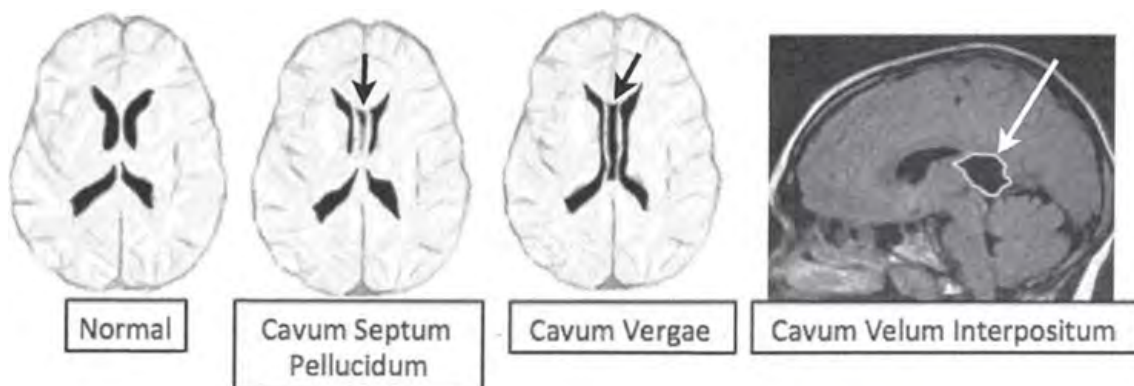
Ventricular Anatomy: Just a quick refresher on this. You have two lateral ventricles that communicate with the third ventricle via the interventricular foramen (of Monroe), which in turn communicates with the fourth ventricle via the cerebral aqueduct. The fluid in the fourth ventricle escapes via the median aperture (foramen of Magendie), and the lateral apertures (foramen of Luschka). A small amount of fluid will pass downward into the spinal subarachnoid spaces, but most will rise through the tentorial notch and over the surface of the brain where it is reabsorbed by the arachnoid villi and granulations into the venous sinus system. Blockage at any site will cause a noncommunicating hydrocephalus. Blockage of reabsorption at the villi / granulation will also cause a noncommunicating hydrocephalus.



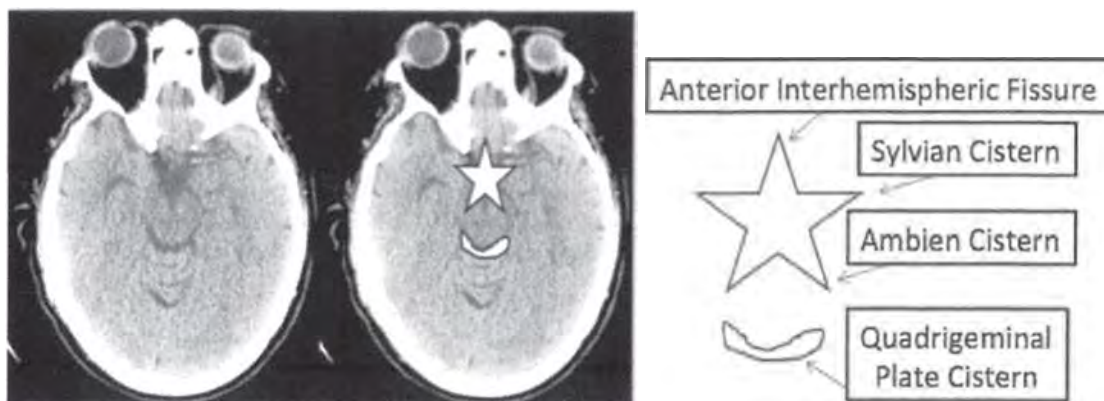
Arachnoid Granulations: These are regions where the arachnoid projects into the venous system allowing for CSF to be reabsorbed. They are hypodense on CT (relative to CSF), and usually round or oval. This round shape helps distinguish them from clot in a venous sinus (which is going to be linear). These things can scallop the inner table (probably from CSF pulsation)

Cavum Variants:

- *Cavum Septum Pellucidum* - Seen in 80% of term infants and 15% of adults. Rarely, can dilate and cause obstructive hydrocephalus
- *Cavum Vergae* - A posterior continuation of the cavum septum pellucidum (never exists without a cavum septum pellucidum)
- *Cavum Velum Interpositum* - Extension of the quadrigeminal plate cistern to foramen of Monro. Seen on sagittal as above the 3rd ventricle and below the fornices.



Basal cisterns: The basal cisterns are good for two things (1) looking for mass effect and (2) anatomy questions. Some people say the suprasellar cisterns look like a pentagon. The five corners of the star lend themselves easily to multiple choice questions: the top of the star is the interhemispheric fissure, the anterior points are the sylvian cisterns, and the posterior points are the ambient cisterns. The quadrigeminal plate looks like a smile.



Brain Development:

Brain Myelination: The baby brain has essentially the opposite signal characteristics as the adult brain. **The T1 pattern of a baby, is similar to the T2 pattern of an adult.** The T2 pattern of a baby, is similar to the T1 pattern of an adult. This appearance is the result of myelination changes. The process of myelination occurs in a predetermined order, and therefore lends itself easily to multiple choice testing. The basic concept to understand first is that immature myelin has a higher water content relative to mature myelin and therefore is brighter on T2 and darker on T1. During the maturation process water will decrease, and fat (brain cholesterol and glycolipids) will increase. Therefore mature white matter will be brighter on T1 and darker on T2.

Immature Myelin

High Water, Low Fat

Relatively T1 dark, T2 bright

Mature Myelin

Low Water, High Fat

Relatively T1 bright, T2 dark

As a point of highly testable trivia: the T1 changes precede the T2 changes (adult T1 pattern seen around age 1, adult T2 pattern seen around age 2). Should be easy to remember (*1 for T1, 2 for T2*).

Order of progression: Just remember, inferior to superior, posterior to anterior, central to peripheral, and sensory fibers prior to motor fibers. The testable trivia, is that **the subcortical white matter is the last part of the brain to myelinate**, with the occipital white matter around 12 months, and the frontal regions finishing around 18 months. The “terminal zones” of myelination occur in the subcortical frontotemporoparietal regions - finishing around 40 months. Another high yield piece of testable trivia is that the **brainstem, and posterior limb of the internal capsule are normally myelinated at birth.**

Brain Myelination Pattern



Inferior to Superior, Posterior to Anterior

Corpus Callosum: I'll touch on this again in the developmental/congenital section but it's high yield enough to repeat. **The corpus callosum forms front to back (then rostrum last).** Therefore hypoplasia of the corpus callosum is usually absence of the splenium (with the genu intact).

High Yield Points Regarding Brain Development

Myelination Occurs Inferior to Superior, and Posterior to Anterior

The Corpus Callosum Forms Front to Back (with the rostrum last)

Both the Anterior and Posterior Pituitary are Bright at Birth (posterior only bright around 2 months - 2 years)

Calverial Bone Marrow will be active (T1 hypointense) in young kids and fatty (T1 hyperintense) in older kids

The sinuses form in the following order: Maxillary, Ethmoid, Sphenoid, and Frontal Last

Brain Iron increases with age (globus pallidus darkens up).

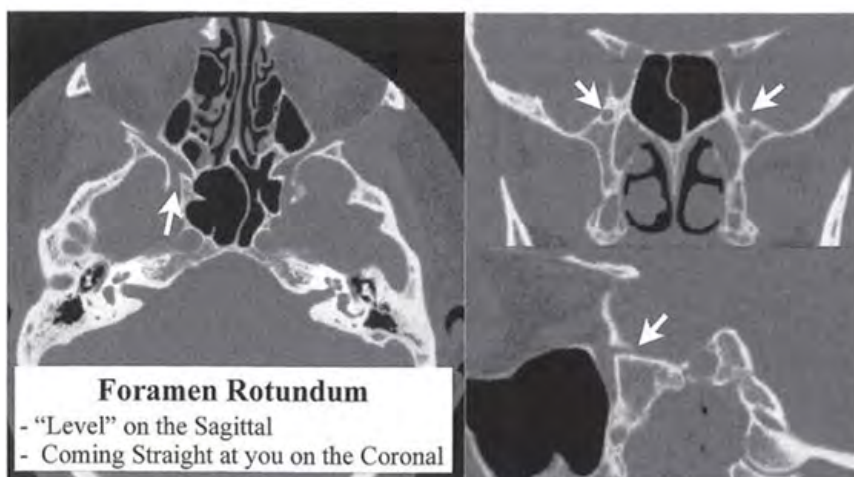
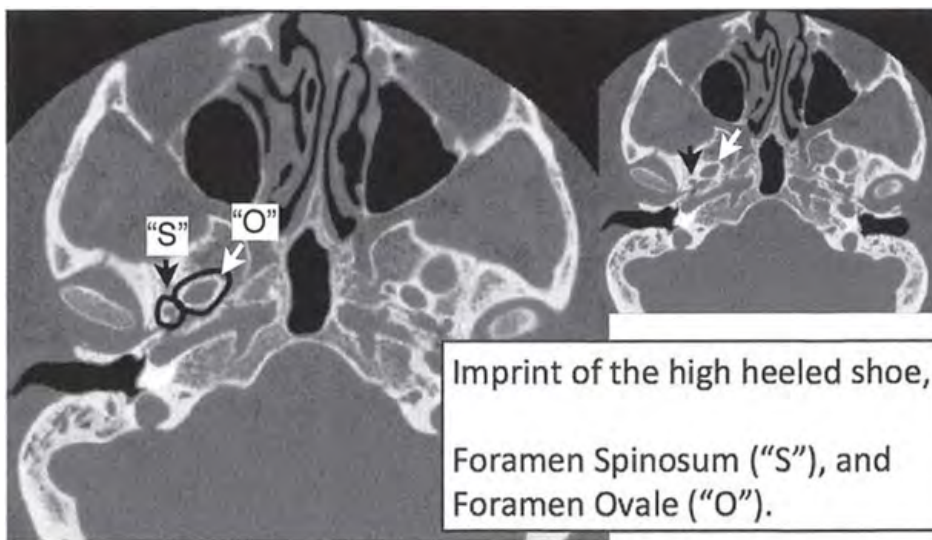
Bony Anatomy:

Skull Base: The most likely multiple choice questions regarding the skull base are anatomy questions. Specifically, the "*what goes where?*" question is very easy to write.

Foramen	Contents
Foramen Ovale	CN V3, and Accessory Meningeal Artery
Foramen Rotundum	CN V2 ("R2V2"),
Superior Orbital Fissure	CN 3, CN 4, CNV1.CN6
Inferior Orbital Fissure	CN V2
Foramen Spinosum	Middle Meningeal Artery
Jugular Foramen	Jugular Vein, CN 9, CN 10, CN 11
Hypoglossal Canal	CN12
Optic Canal	CN 2, and Ophthalmic Artery

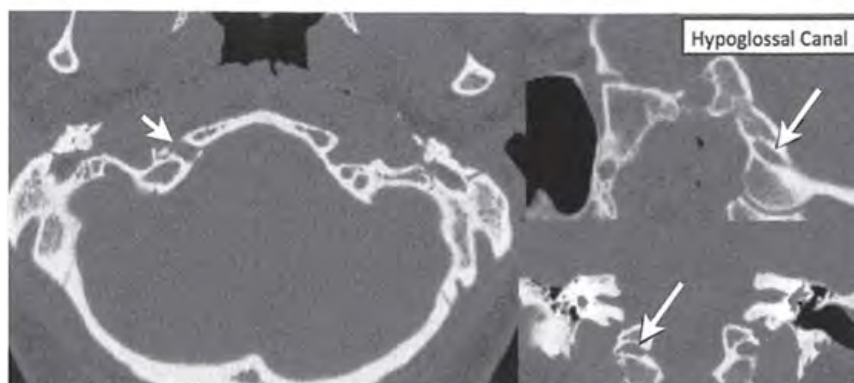
Remember, that they don't have to show you the hole in the axial plane. They can be sneaky and show it in the coronal or sagittal planes. In fact, showing foramen rotundum in the coronal and sagittal planes is a very common sneaky trick.

With regard to the relationship between **spinosum** and **ovale**, I like to think of this as the foot print a woman's high heeled shoe might make in the snow, with the oval part being ovale, and the pointy heel as spinosum.



With regard to **Rotundum**, think about it as being totally level or horizontal on the sagittal view.

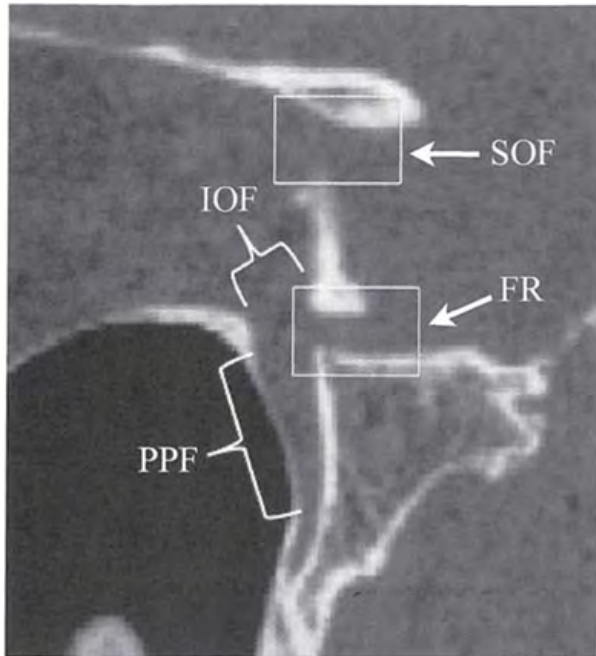
On the coronal view, it looks like you are staring into a gun barrel.



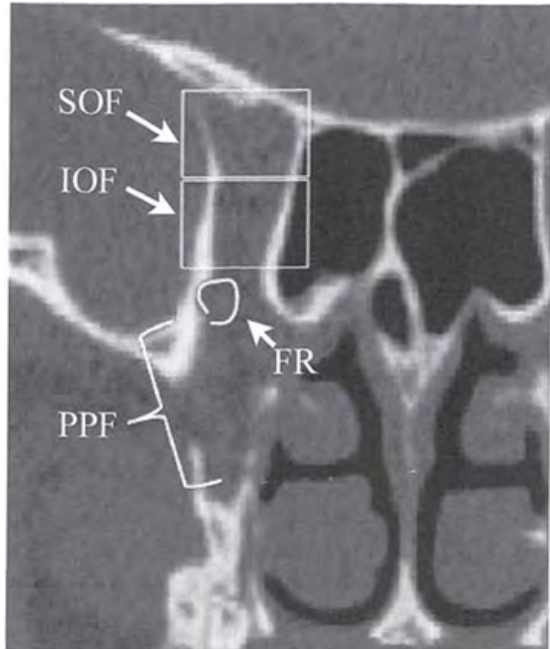
The **hypoglossal canal** is very posterior and inferior.

This makes it unique as a skull base foramen.

The relationship between the superior orbital fissure (SOF), the inferior orbital fissure (IOF), foramen rotundum (FR), and the pterygopalatine fossa (PPF) is an important one, that can really lead to some sneaky multiple choice questions (mainly what goes through what). I've attempted to outline this relationship on both sagittal and coronal views.



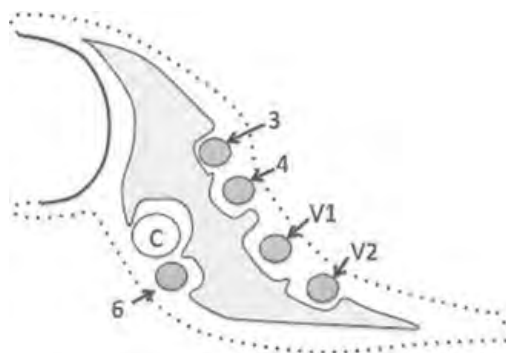
Sagittal



Coronal

Cavernous Sinus: - The question is going to be, what's in it (probably asked as what is NOT in it). CN3, 4, CN VI, CNV2, CN6, and the carotid. **CN2 and CN V3 do NOT run through it.**

The only other anatomy trivia I can think of is that CN6 runs next to the carotid, the rest of the nerves are along the wall. This is why you can get lateral rectus palsy earlier with cavernous sinus pathologies.



Skull fusion (Craniosynostosis): The craniosynostoses are discussed in more detail in the congenital section. I'll briefly touch on what is normal. The sutures exist to allow for rapid brain growth over the first few years of life. The brain will double in size within the first 6 months, and double again by the second year of life. The majority of skull growth occurs by age 3, at which time most of the sutures will fuse. Some, like the petro-occipital, will remain open into adulthood. When they fuse too early that causes a problem. The long Latin / French sounding word that goes along with that fusion problem makes for a good multiple choice test question (more on this later).

Vascular Anatomy:

Vascular anatomy can be thought of in four sections. (1) The branches of the external carotid (most commonly tested as the order in which they arise from the common carotid). (2) Segments of the internal carotid, with pathology at each level and variants. (3) Vertebral artery, with pathology. (4) Circle of Willis, with pathology and variants.

What are the branches of the external carotid ?

- *Some Administrative assistants Like Fucking Over Poor Medical Students*
 - Superior Thyroid
 - Ascending Pharyngeal
 - Lingual
 - Facial
 - Occipital
 - Posterior Auricular
 - Maxillary
 - Superficial Temporal

Anterior Circulation (Carotids):

CI (Cervical): 4 main pathologies of interest at this level:

- Atherosclerosis: The origin is a very common location
- Dissection: Can be spontaneous (women), and in Marfan's or Ehlers-Danlos, and result in a partial Horner's (ptosis and miosis), followed by MCA territory stroke.
- Can have a retropharyngeal course and get "drained" by ENT accidentally.
- Pharyngeal infection may cause pseudoaneurysm at this level.

C2 (Petrous): - Not much goes on at this level. Sometimes aneurysms (which can be surprisingly big).

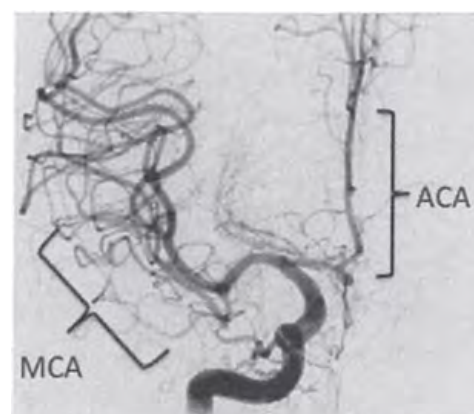
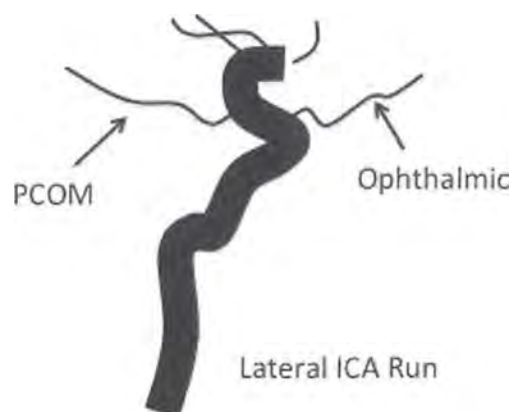
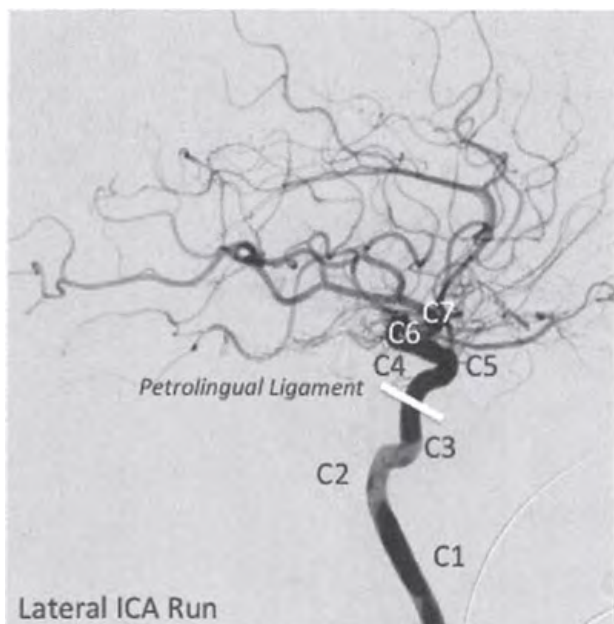
C3 (Lacerum): Not much here as far as vascular pathology. The anatomic location is important to neurosurgeons for exposing Meckel's cave via a transfacial approach.

C4 (Cavernous): Aneurysms here are strongly associated with hypertension. This segment is affected by multiple pathologies including the development of cavernous - carotid fistula.

C5 (Clinoid): An aneurysm here could compress the optic nerve and cause blindness.

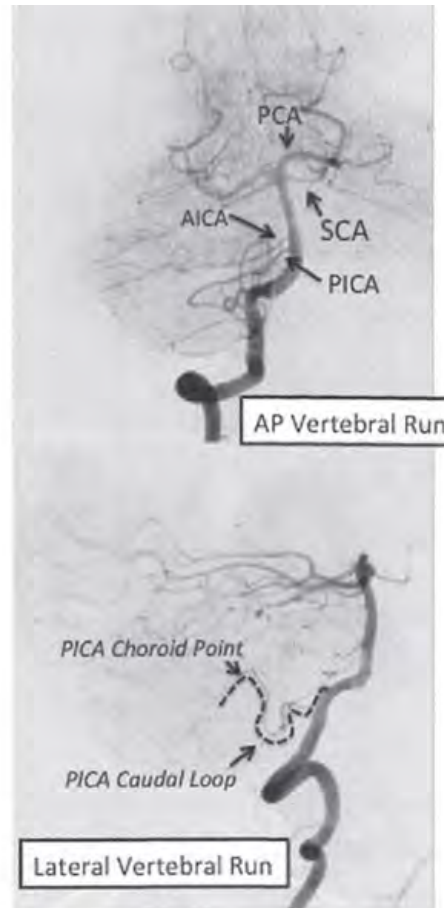
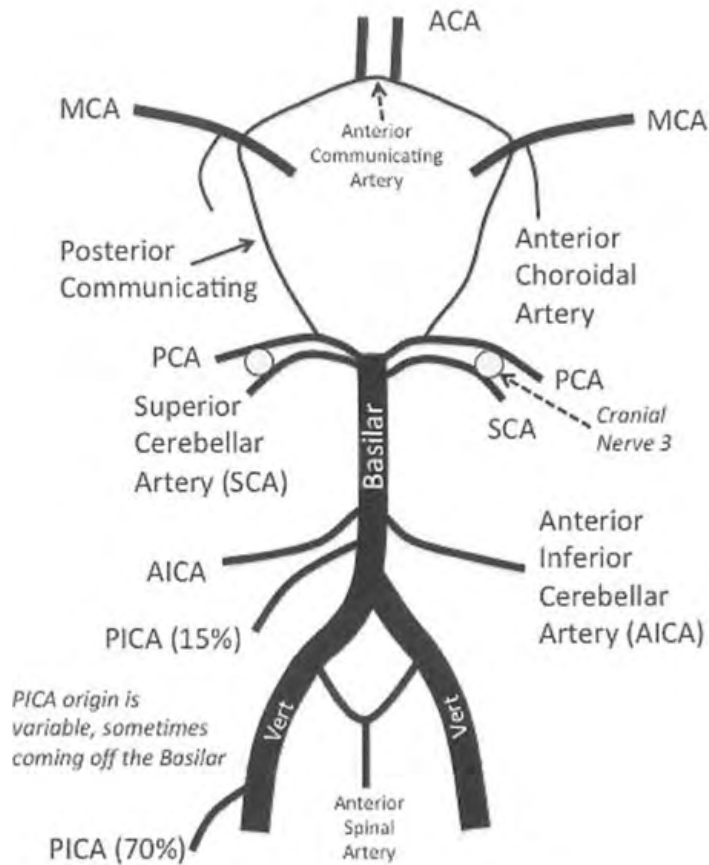
C6 (Ophthalmic - Supraclinoid): Common site for aneurysm formation. **Origin at the "dural ring" is a buzzword** for this artery.

C7 (Communicating - terminal): An aneurysm here may compress CN III and present with a palsy.



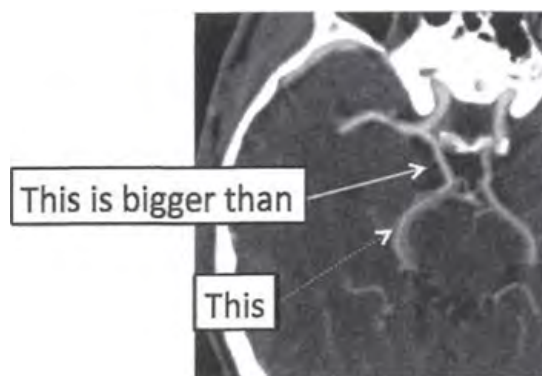
APICA Run

Posterior Circulation:



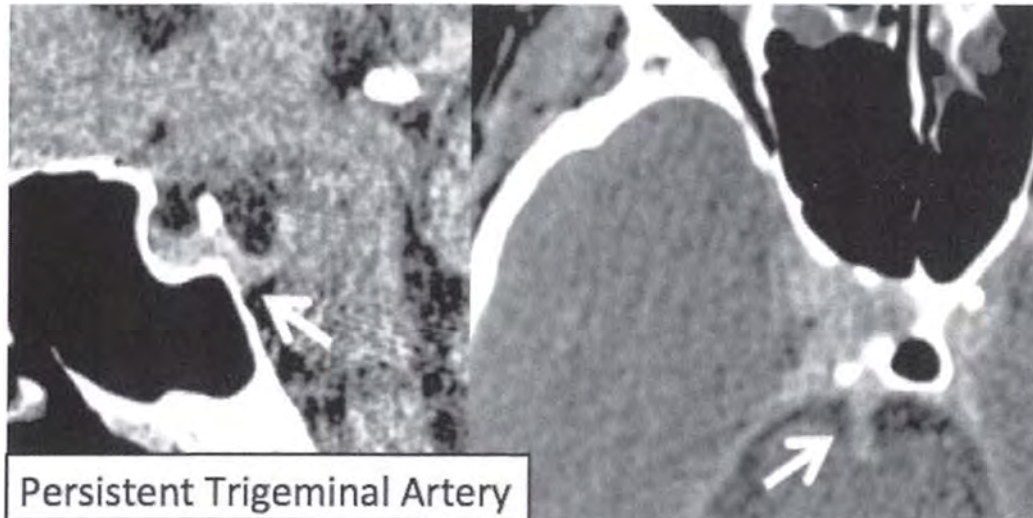
Vascular Variants:

Fetal Origin of PCA: Most common vascular variant (probably) - seen in up to 30% of general population. The term "fetal PCOM" is typically used to refer to a situation where the PCOM is larger (or the same size) as PI. Another piece of trivia is that *anatomy with a fetal PCA has the PCOM superior / lateral to CN3 (instead of superior / medial - in normal anatomy).*

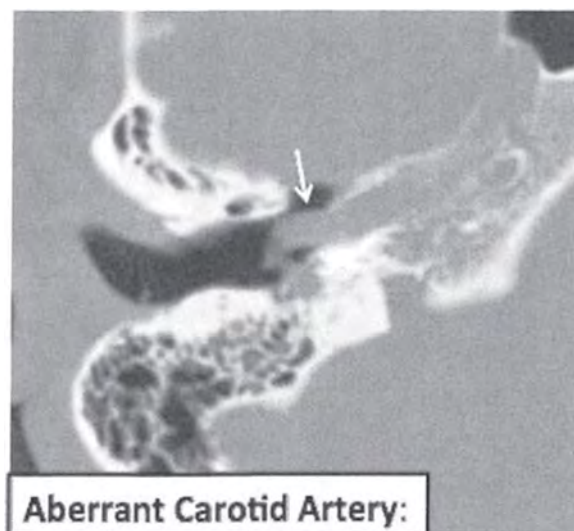


Fetal PCOM

Persistent Trigeminal Artery: Persistent fetal connection between the cavernous ICA to the basilar artery. A characteristic “*tau sign*” on Sagittal MRI has been described. It **increases the risk of aneurysm** (anytime you have branch points).



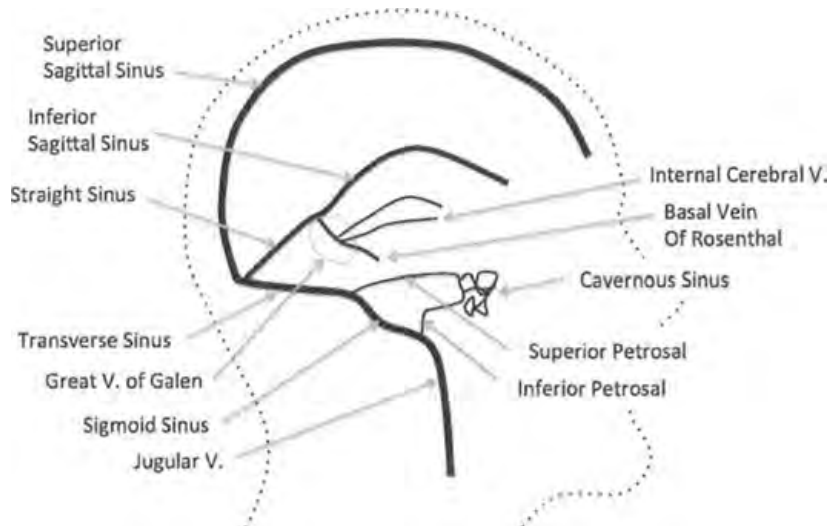
Aberrant Carotid Artery: Typically represents an enlarged inferior tympanic artery anastomosis with an enlarged caroticotympanic artery (with underdevelopment of the cervical ICA). This vessel courses through the tympanic cavity and joins the horizontal carotid canal. It can cause pulsatile tinnitus. The oldest trick in the book is to try and fool you into calling it a paraganglioma. Don't biopsy it!



Venous Anatomy:

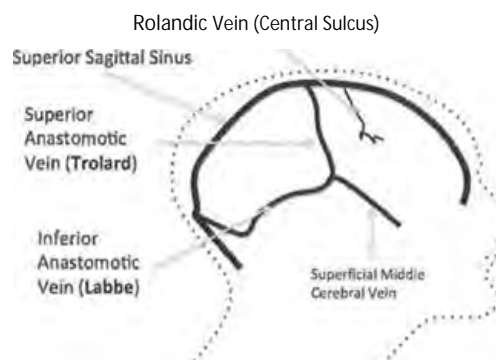
You can ask questions about the venous anatomy in roughly three ways (1) what is it - on a picture, (2) what is a deep vein vs what is a superficial vein, (3) trivia.

What is it?



Superficial vs Deep

<i>Superficial</i>	<i>Deep</i>
Superior Cerebral Veins	Basal Vein of Rosenthal
Superior Anastomotic Vein of Trolard	Vein of Galen
Inferior Anastomotic Vein of Labbe	Inferior Petrosal Sinus
Superficial Middle Cerebral Veins	



Trolard = Top
Labbe = Lower

Venous Trivia:

Collateral Pathways: The dural sinuses have accessory drainage pathways (other than the jugular veins) that allow for connection to extracranial veins. These are good because they can help regulate temperature, and equalize pressure. These are bad because they allow for passage of sinus infection / inflammation, which can result in venous sinus thrombosis.

Inverse Relationship: There is a relationship between the Vein of Labbe, and the Anastomatic Vein of Trolard. Since these dudes share drainage of the same territory, as one gets large the others get small.

Sounds Latin or French: As a general rule, anything that sounds Latin or French has an increased chance of being on the test.

- *Vein of Labbe:* Large draining vein, connecting the superficial middle vein and the transverse sinus
- *Vein of Trolard:* Smaller (usually) vein, connecting the superficial middle vein and sagittal sinus
- *Basal vein of Rosenthal:* Deep vein that passes lateral to the midbrain through the ambient cistern and drains into the vein of Galen. Its course is similar to the PCA.
- *Vein of Galen:* W\g vein ("great") formed by the union of two internal cerebral veins.

Trivia you need to remember about Neuro Anatomy

- ***First Order Trivia:***
 - "What is it? " Style questions are most likely; with possibilities including CTA, MRA, or Angiograms. Considering when the people writing the questions trained, angiograms are probably the most likely.
 - "What goes through there? " Neuro foramina
 - "What doesn't?" Style questions - CN 2 and CN V3 don't go through the cavernous sinus.
- ***Second Order Trivia:***
 - CN 3 Palsy - Think Posterior Communicating Artery Aneurysm
 - CN 6 Palsy - Think increased ICP

Misc Brain Conditions

Monro-Kellie Doctrine:

The Monro-Kellie doctrine is the idea that the head is a closed shell, and that the three major components: (1) brain, (2) blood - both arterial and venous, and (3) CSF, are in a state of dynamic equilibrium. As the volume of one goes up, the volume of another must go down.

Intracranial Hypotension: If you are leaking CSF, this will decrease the overall fixed volume, the volume of venous blood will increase to maintain the equilibrium. The result is meningeal engorgement (enhancement), distention of the dural venous sinuses, prominence of the intracranial vessels and engorgement of the pituitary. The development of subdural hematoma and hygromas is also a classic look (again, compensating for lost volume).

Idiopathic Intracranial Hypertension (Pseudotumor Cerebri): Classic scenario of a fat middle aged women with a headache. Etiology is not well understood (making too much CSF, or not absorbing it correctly). It has a lot of associations (hypothyroid, cushings, vitamin A toxicity). The findings follow the equilibrium idea. With increased CSF the ventricles become slit like, the pituitary shrinks (partially empty sella), and the venous sinuses appear compressed. You can also have the appearance of vertical tortuosity of the optic nerves and flattening of the posterior sclera.

Edema:

Cytotoxic: This type of edema can be thought about as intracellular swelling, secondary to malfunction of the Na/K pump. It tends to favor the gray matter, and **looks like loss of the gray-white differentiation**. This is classically **seen with stroke** (or trauma), and is why early signs of stroke involve loss of the GM-WM interface.

Vasogenic: This type of edema is extracellular, secondary to disruption of the blood brain barrier. It looks like **edema tracking through the white matter** (which is less tightly packed than the gray matter). This is classically **seen with tumor and infection**. A response to steroids is characteristic of vasogenic edema.

Hydrocephalus

You can classify these things as non-obstructive or obstructive; and sub-classify obstructive into non-communicating or communicating.

Obstructive.

Non-Communicating: The obstruction involves the ventricular system. You are going to have some dilated ventricles and some normal sized (depending on the level of the obstruction). There are 3 main causes:

- (1) **Aqueductal Stenosis** - The lateral ventricles and third ventricle will be big (the fourth ventricle will be normal). This is the **MOST COMMON cause of congenital obstructive hydrocephalus**. It can also be seen in adults as an acquired pathology (but for the purpose of multiple choice, this is a newborn). The congenital types are usually from a web or diaphragm (sometimes gliosis). The acquired types are either from compression (tectal plate glioma, pineal tumor), or intrinsic from ventriculitis or SAH.
- (2) **Intraventricular Mass** - The classic example is the colloid cyst of the third ventricle which can obstruct the foramen of Monro and cause sudden death / thunderclap headache.
- (3) **Outlet Obstruction of the 4th Ventricle** - This can mimic a communicating hydrocephalus, as all ventricles will be dilated. A Dandy Walker malformation can do this. Alternatively, you can acquire this with basilar meningitis or post hemorrhagic ependymitis.

Communicating: This is an obstruction at the level of villi / granulation, blocking reabsorption. All the ventricles will be dilated (25% of the time the fourth ventricle is normal). There are 4 main causes.

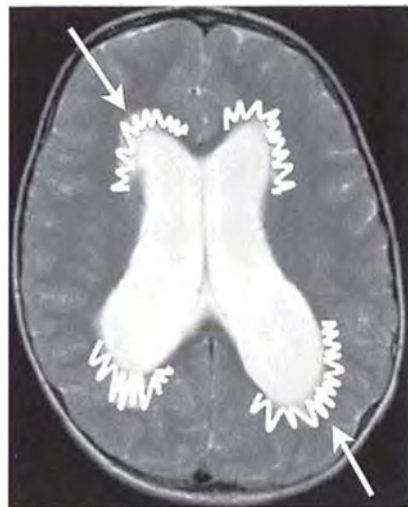
- **Normal Pressure Hydrocephalus:** It's not well understood - and idiopathic. The buzz-phrase is "ventricular size out of proportion to atrophy." The frontal and temporal horns of the lateral ventricles are the most affected. "Upward bowing of the corpus callosum" is another catch phrase. On MR1 you may see transependymal flow and/or a flow void in the aqueduct and 3rd ventricle. The step 1 trivia is "wet, wacky, and wobbly" - describing the clinical triad of urinary incontinence, confusion, and ataxia. This is treated with surgical shunting.
- **Blood, Pus, and Cancer** - Anything that plugs up the villi - the three most common causes being SAH, Meningitis, and Carcinomatous Meningitis.

Non- Obstructive.

This is sort of a trick question, with the only answer being something that produces CSF. The only answer you need to know is Choroid Plexus Papilloma (discussed in detail in the tumor section).

Quiz: Is transependymal flow seen more with acute hydrocephalus or chronic hydrocephalus?

Answer: Acute.



Brain Herniation

Subfalcine Herniation: This is just a fancy way of saying midline shift (deviation of ipsilateral ventricle and bowing of the falx). The trivia to know is that the ACA may be compressed, and can result in infarct.

Descending Transtentorial Herniation: The uncus and hippocampus herniated through the tentorial incisura. Effacement of the ipsilateral suprasellar cistern occurs first:

Things to know:

- Perforating basilar artery branches get compressed resulting in “*Duret Hemorrhages*” - classically located in the midline at the pontomesencephalic junction (in reality they can also effect cerebellar peduncles).
- CN3 gets compressed between the PCA and Superior Cerebellar Artery causing ipsilateral pupil dilation and ptosis
- “*Kernohan’s Notch / Phenomenon*” - The midbrain on the tentorium forming an indentation (notch) and the physical exam finding of ipsilateral hemiparesis - which Neurologist’s call a “*false localizing sign.* ” Of course, localization on physical exam is stupid in the age of MRI, but it gives Neurologists a reason to carry a reflex hammer and how can one fault them for that.

Ascending Transtentorial Herniation: Think about this in the setting of a posterior fossa mass. The vermis will herniate upward through the tentorial incisura often resulting in severe obstructive hydrocephalus.

Things to know

- The “*Smile*” of the quadrigeminal cistern will be flattened or reversed
- “*Spinning Top*” is a buzzword, for the appearance of the midbrain from bilateral compression along its posterior aspect
- Severe hydrocephalus (at the level of the aqueduct).

Cerebellar Tonsil Herniation: Can be from severe herniation after downward transtentorial herniation). Alternatively, if in isolation you are thinking more along the lines of Chiari (Chiari I = 1 tonsil 5mm, or both tonsils 3mm).

Neuro-Degenerative / Toxic Metabolic

Multiple Sclerosis:

By far the most common acquired demyelinating disease. Usually affects women 20-40. As a point of trivia in children there is no gender difference. There are multiple types with the relapsing-remitting form being by far the most common (85%). Clinical history of “separated by time and space” is critical. Findings are the classic T2/FLAIR oval and periventricular perpendicularly oriented lesions. Involvement of the calloso-septal interface is 98% specific for MS (and helps differentiate it from vascular lesions and ADEM). In children the posterior fossa is more commonly involved. Acute demyelinating plaques should enhance and restrict diffusion (on multiple choice tests and occasionally in the real world). Brain atrophy is accelerated in MS.

You can sometimes get a big MS plaque that looks like a tumor. **It will ring enhance but classically incomplete** (*like a horseshoe*), with a leading demyelinating edge. Solitary spinal cord involvement can be seen but it usually is seen in addition to brain lesions. The cervical spine is the most common location in the spine (65%). Spinal cord lesions tend to be peripherally located.

Multiple Sclerosis Variants:

ADEM (Acute Disseminated Encephalomyelitis): Typically presents in childhood or adolescents, after a viral illness or vaccination. Classically has multiple LARGE T2 bright lesions, which enhance in a nodular or ring pattern (open ring). Lesions **do NOT involve the calloso-septal interface**.

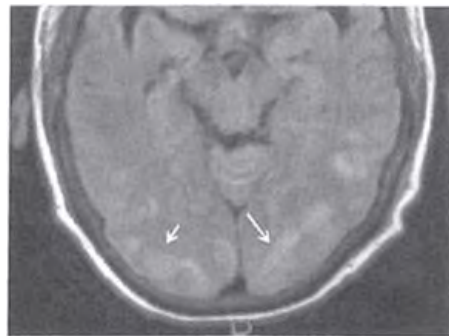
Acute Hemorrhagic Leukoencephalitis (Hurst Disease): This a fulminant form of ADEM with massive brain swelling and death. The hemorrhagic part is only seen on autopsy (not imaging).

Devics (neuromyelitis optica): Transverse Myelitis + Optic Neuritis.

Marburg Variant: Childhood variant that is fulminant and terrible leading to rapid death. It usually has a febrile prodrome.

Toxic /Metabolic

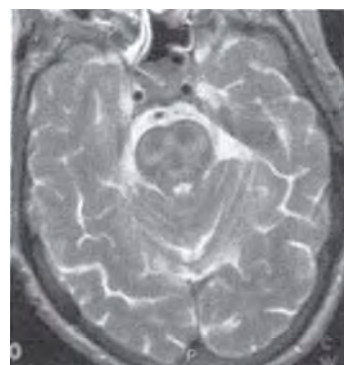
PRES (Posterior Reversible Encephalopathy Syndrome): Seen with hypertension or chemotherapy. Features include asymmetric cortical and subcortical white matter edema (usually in parietal occipital regions). PRES does NOT restrict on diffusion (helps tell you it's not a stroke).



PRES - T2/FLAIR High Signal

Radiation-Induced Demyelination: Seen as T2 bright areas and atrophy corresponding to the radiation portal. Can be seen with hemosiderin deposition, and mineralizing microangiopathy (calcifications involving the basal ganglia and subcortical white matter).

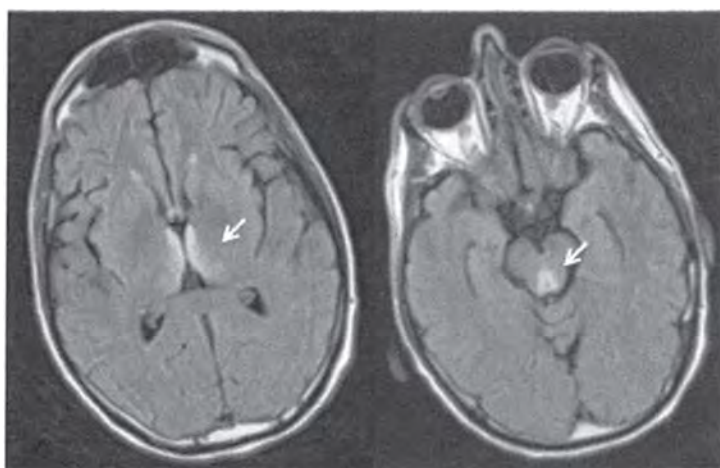
Osmotic Demyelination Syndrome (CPM): Seen with rapid correction of sodium (usually in a drunk). Usually T2 bright in the central pons (sparing the periphery). Can also have an extra-pontine presentation involving the basal ganglia, external capsule, amygdala, and cerebellum.



CPM - T2 Bright Central Pons

Wernicke Encephalopathy:
Caused by thiamine deficiency. Just think contrast enhancement of the **mammillary bodies** (seen more in alcoholics).

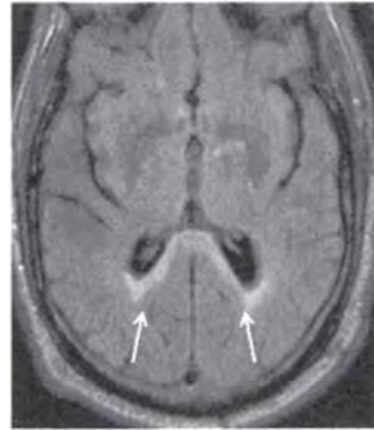
Additionally, think increased T2/FLAIR signal in the bilateral medial thalamus and periaqueductal gray.



*Wernicke
High Signal in Medial Thalamus and Periaqueductal Gray*

CNS Findings Secondary to Drugs or Toxins:

- **Carbon Monoxide:** CT Hypodensity / T2 Bright Globus Pallidus {*carbon monoxide causes “globus ” warming*}.
- **Alcohol:** Brain atrophy , especially the cerebellar vermis.
- **Marchiafava-Bignami:** Seen in drunks. Swelling and T2 bright signal affecting the **corpus callosum** (typically beginning in the body, then genu, and lastly splenium). Will involve the central fibers and spare the dorsal and ventrals fibers (called a “**sandwich sign**” on sagittal imaging).



Marchiafava-Bignami
- High T2/FLAIR in the Corpus Collosum

- **Methanol:** Optic nerve atrophy, hemorrhagic **putaminal** and subcortical white matter necrosis

Post-Radiation: There is a latent period, so imaging findings don't typically show up for about two months post therapy.

- *Whole Brain Radiation* changes are typically T2 bright in the periventricular white matter, sparing the subcortical regions early on. Peripheral extension to the subcortical regions occurs later.
- *Localized Radiation:* Usually we are talking about severe focal edema with mass effect and enhancement. Differentiation from residual tumor can be a sneaky sneaky thing, and MR perfusion may be useful in differentiating.

Post Chemotherapy: You will have T2 effects acutely in the white matter, that can progress to atrophy. Enhancement or mass effect is rare unless it is very severe. Children receiving both radiation and chemotherapy can sometimes develop calcifications - “mineralizing microangiopathy.”

Disseminated necrotizing leukoencephalopathy: Severe white matter changes, which demonstrate ring enhancement, classically seen with leukemia patients undergoing radiation and chemotherapy. This is bad news and can be fatal.

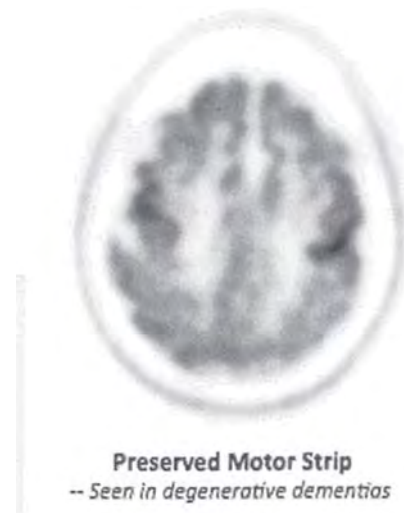
Neurodegenerative Disorders:

You can do dementia imaging with a variety of imaging modalities, including CT and MRI for structure, and FDG PET and SPECT for function. **Pearl: On FDG PET the motor strip is always preserved in dementia.**

Mimics:

Depression can mimic Frontotemporal Dementia.

Lymes, HIV, and Vasculitis can mimic Vascular Dementia

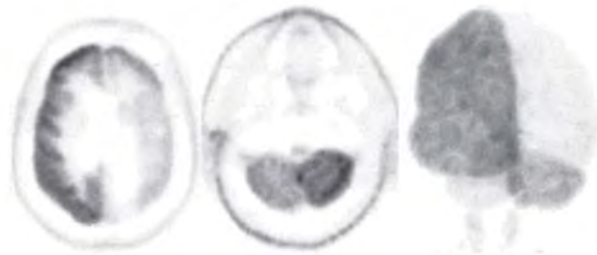


Alzheimer Disease: Most common cause of dementia. Most likely question is **hippocampal atrophy** (which is first), and out of proportion to the rest of the brain atrophy. They could ask temporal horn atrophy > 3mm, which is seen in more than 65% of cases.

Multi-infarct Dementia: This is the second most common cause of dementia. Cortical infarcts and lacunar infarcts are seen on MRI. Most likely to be shown as a PET-FDG case, demonstrating multiple scattered areas of decreased activity

Crossed Cerebellar Diaschisis (CCD):

Depressed blood flow and metabolism affecting the cerebellar hemisphere after a contralateral supratentorial insult (infarct, tumor resection, radiation). Creates an Aunt Minnie Appearance:



Crossed Cerebellar Diaschisis

Dementia with Lewy Bodies: This is the third most common cause of dementia (second most common neurodegenerative), with a very similar clinical picture to the dementia seen with Parkinsons, with the major difference being that in DLB, the dementia comes first. The hippocampi remain normal in size and you have some decreased FDG uptake in the lateral occipital cortex, with sparing of the mid posterior cingulate gyrus (**Cingulate Island Sign**).

Binswanger Disease: This is a subcortical leukoencephalopathy that affects older people (55 and up), strongly associated with HTN. It's basically a form of *small vessel vascular dementia*. It classically spares the subcortical U fibers.

FDG PET - Brain

Alzheimer	Low posterior temporoparietal cortical activity	Identical to Parkinson Dementia
Multi Infarct	Scattered areas of decreased activity	<i>Lyme, HIV, and Vasculitis are mimics</i>
Dementia with Lewy Bodies	Low in lateral occipital cortex	Preservation of the mid posterior cingulate gyrus (Cingulate Island Sign)
Picks / Frontotemporal / Depression	Low frontal lobe	<i>Depression is a mimic</i>
Huntingtons	Low activity in caudate nucleus and putamen	

Infections

My idea for discussing intracranial infections is to think of a few “testable” scenarios. The neonatal infections, the infections related to HIV, the “characteristic” infections, and lastly meningitis and cerebral abscess.

Neonatal Infections:

We are talking about TORCH infections. The first critical thing is that they only really matter in the first two trimesters (doesn't cause as much harm in third trimester). Calcifications and microcephaly are basically present in all of them.

Here are some high yield points regarding the TORCH infections:

CMV: This is the **most common TORCH** (by far!, it's 3x more common than Toxo - which is the second most common). It likes to affect the germinal matrix and causes periventricular tissue necrosis. The result is the most likely test question = **Periventricular calcifications**. Another high yield piece of trivia is that of all the TORCHs **CMV has the highest association with polymicrogyria**.

Toxoplasmosis: This is the second most common TORCH. It's seen in women who clean up cat shit. The calcification pattern is more random, and affects the basal ganglia (like most other TORCH infections). The frequency is increased in the 3rd trimester (but only causes a problem in the first two). The most likely test question = **hydrocephalus**.

Rubella: Less common because of vaccines. Calcifications are less common than in other TORCHs. On MR, focal high T2 signal might be seen in white matter (related to vasculopathy and ischemic injury).

HSV: As a point of trivia, it's usually HSV-2 in 90% of cases. Unlike adults, the virus does not primarily affect the limbic system but instead affects the endothelial cells resulting in **thrombus and hemorrhagic infarction** with resulting encephalomalacia and atrophy.

HIV: This is not a TORCH but does occur during pregnancy, at delivery, or through breast feeding. **Brain atrophy predominantly in the frontal lobes** is the main testable piece of trivia. You may also have faint basal ganglia enhancement seen on CT and MRI preceding the appearance of basal ganglia calcification.

CNS TORCH - What you need to remember

- CMV = *Most Common, Periventricular Calcifications, Poly microgyria*
- Toxo = *Hydrocephalus, Basal Ganglia Calcifications*
- Rubella = *Vasculopathy/Ischemia. High T2 signal -Less Calcifications*
- HSV = *Hemorrhagic Infarct, and lead to bad encephalomalacia (hydranencephaly)*
- HIV = *Brain Atrophy in frontal lobes*

Infections Immunosuppressed Patients Get (people with AIDS)

The most common opportunistic infection in patients with AIDS is toxo. The most common fungal infection (in people with AIDS) is Cryptococcus. Two other infections worth talking about are JC Virus, and CMV.

HIV Encephalitis: I'll lead with the encephalitis people with AIDS get. This is actually pretty common and affects about 50% of AIDS patients. Usually we are talking about a situation with a CD4 < 200. What you are going to see is **symmetric** increased T2 / FLAIR signal in the deep white matter. **T1 will be normal.** The lesions will not enhance. There may be associated brain atrophy. These tend to **spare the subcortical U-fibers** (*PML will involve them*).

Progressive Multifocal Leukoencephalopathy (PML): This is caused by the JC virus. We are talking about a situation with a CD4 less than 50. The imaging manifestations are a single or multiple scattered hypodensities, with corresponding **T1 hypointensity** (remember HIV was T1 normal), and **T2/FLAIR hyperintensities out of proportion to mass effect - buzzword.** The lesions have a predilection for the **subcortical U-fibers**. The **asymmetry** of these lesions helps differentiate them from HIV Encephalitis.



HIV Encephalitis

•Symmetric, and Spare Cortical U Fibers



PML

-Asymmetric, and Involves Cortical U Fibers



WTF are these "U Fibers" ?

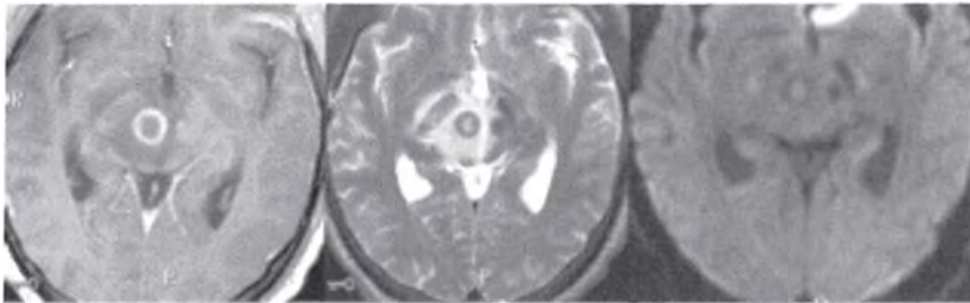
They are the fibers under the cortex, that look like "U"s. They come up a lot, as being spared or not spared.



CMV: Think about brain **atrophy**, periventricular hypodensities (that are T2/FLAIR bright), and **ependymal enhancement**.

Cryptococcus: The most common fungal infection in AIDS. The most common presentation is meningitis that affects the base of the brain (leptomeningeal enhancement). The most likely way this will be shown on a multiple choice exam is **dilated perivascular spaces filled with mucoid gelatinous crap (these will not enhance)**. The second most likely way this will be shown is lesions in the basal ganglia "cryptococcomas" - these are T1 dark, T2 bright, and may ring enhance.

Toxo: Most common opportunistic infection in AIDS. Classically we are talking about T1 dark, T2 bright, ring enhancing (when larger than 1cm) lesions. These guys will NOT show restricted diffusion. Just think “ring enhancing lesion, with LOTS of edema.” Most likely test question is that Toxo is Thallium Cold, and Lymphoma is thallium hot.



TOXO: Ring Enhancing. Lots of Edema, Not Restricted

Wait -I thought abscess restricted diffusion?

Typical abscess does. However, atypical infections like Toxo or fungal don't always do this, and showing that it does NOT restrict might be a sneaky way to test this.

Toxo	Lymphoma
Ring Enhancing	Ring Enhancing
Hemorrhage more common after treatment	Hemorrhage less common after treatment
Thallium Cold	Thallium HOT
PET Cold	Pet Hot
MR Perfusion: Decreased CBV	MR Perfusion: Increased (or Decreased) CBV

Summary:

AIDS Encephalitis	PML	CMV	Toxo	Cryptococcus
Symmetric T2 Bright	Asymmetric T2 Bright	Periventricular T2 Bright	Ring Enhancement	Dilated Perivascular Spaces
	T1 dark	Ependymal Enhancement	Thallium Cold	Basilar Meningitis

The Characteristic Infections:

TB Meningitis: TB meningitis looks just like regular meningitis, except that it has a predilection for the basal cisterns and may have dystrophic calcifications. There may be **enhancement of the basilar meninges with minimal nodularity**. Complications include vasculitis which may result in infarct (more common in children). Obstructive hydrocephalus is common. *Most likely way to show this is enhancement of the basilar meninges (sarcoid can do that too - so it won't be a distractor unless he/she also has hydrocephalus - in which case pick TB).*

HSV: It's HSV 1 in adults and HSV 2 in neonates. I mention that because (1) it seems like testable trivia and (2) they actually have different imaging appearances (as mentioned above type 2 doesn't love the limbic like type 1). For the purpose of multiple choice test you are going to have a swollen (unilateral or bilateral) **medial temporal lobe**, which will be T2 bright. *Earliest sign is actually restricted diffusion* - related to vasogenic edema. This could be tested by asking "what sequence is more sensitive?", with the answer being the **diffusion is more sensitive than T2**. Blooming on gradient means it's bleeding (common in adults, rare in neonate form). Other trivia is that it **sparing the basal ganglia** (distinguishes it from MCA stroke).



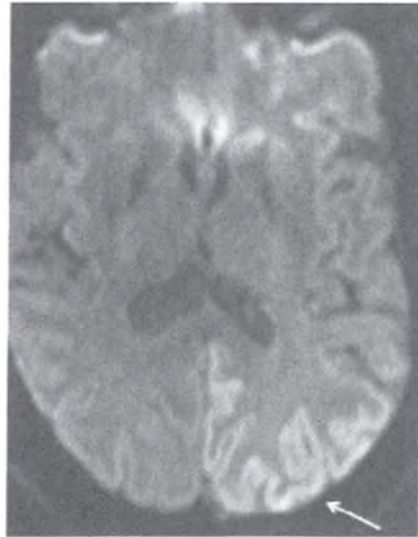
Herpes-
Edema in the temporal lobe

Limbic Encephalitis: Not an infection, but a commonly tested mimic. It is a **paraneoplastic syndrome (usually small cell lung cancer)**, that looks very **similar to HSV**. This could be asked by showing a classic HSV image, but then saying HSV titer negative. The second order question would be to ask for lung cancer screening.

West Nile: Several viruses characteristically involve the basal ganglia (Japanese Encephalitis, Murray Valley Fever, West Nile...), the only one realistically testable is West Nile. We are talking about **T2 bright basal ganglia and thalamus, with corresponding restricted diffusion**. Hemorrhage is sometimes seen.

CJD: There are 3 types: sporadic (80-90%), variant (rare), and familial (10%). Random factoid is that it has a characteristic appearance on EEG, and this “14-3-3” protein assay is a CSF test neurologists order. The imaging features are variable and can be unilateral, bilateral, symmetric, or asymmetric. I want to concentrate on the most likely testable appearances of which I think there are 3.

- (1) *DWI Showing Cortical Gyriform restricted signal* - supposedly diffusion is the most sensitive sign, and the cortex is the most common early site of manifestation.
- (2) *“Hockey Stick Sign / Pulvinar Sign ”* - Restricted diffusion in the dorsal medial thalamus (which looks like a hockey stick), or in the pulvinar
- (3) *A series of MRs or CTs showing rapidly progressive atrophy*



CJD: Gyriform Restricted Diffusion

Neurocysticercosis: Caused by eating pig shit (or undercooked pork). The bug is *taenia solium*. The most common locations of involvement are the subarachnoid space over the cerebral hemispheres, basal cisterns, brain parenchyma, and ventricles (in that descending order). As a point of trivia, involvement of the basal cisterns carries the worst outcome.

It has 4 stages which could be written in the form of a multiple choice questions (that would be really dirty, and therefore likely: •

- Vesicular - thin walled cyst (iso-iso T1/T2 + no edema)
- Colloidal - hyperdense cyst (bright-bright T1/T2 + edema)
- Granular - cyst shrinks, wall thickens (less edema)
- Nodular - small calcified lesion (no edema)

Meningitis and Cerebral Abscess

You can think of meningitis in 4 main categories: bacterial (acute pyogenic), viral (lymphocytic), chronic (TB or Fungal), and non-infectious (sarcoid).

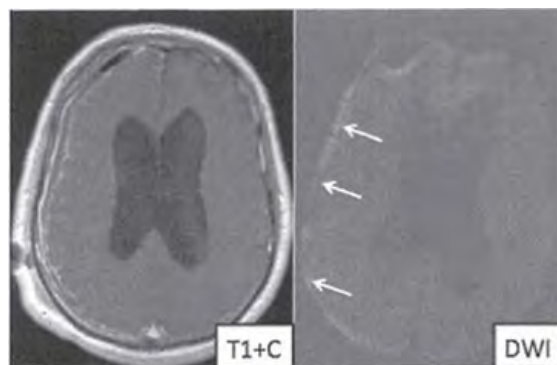
Essentially, we are talking about thick leptomeingeal enhancement, in the appropriate clinical setting. The complications are numerous and include venous thrombosis, vasospasm (leading to the stroke), empyema, ventriculitis, hydrocephalus, abscess etc... and so on and so forth.

Abscess Facts (trivia)

- *DWI - Restricts*
- *MRS - Lactate High*
- *PET FDG - Increased Metabolic*

A very testable piece of trivia is that infants will often get sterile reactive subdurals (much less common than in adults).

Empyema: Can be subdural or epidural (just like blood). Follows the same rules as far as crossing dural attachments (epidurals don't) and crossing the falx (subdurals don't). Subdurals are more common and have more complications relative to epidurals. The vast majority of subdurals are the sequela of frontal sinusitis. The same is true of epidurals with some sources claiming 2/3 of epidurals are secondary to sinusitis. They are often T1 bright and can **restrict on diffusion**.



Subdural Empyema: Dural Enhancement, Restricts

Intraaxial Infections; We are talking Cerebritis, Abscess, and Ventriculitis:

Again lots of causes via direct spread, or hematogenous spread. The causes worth thinking about include right to left shunts, and pulmonary AVMs. Cerebritis is the early form of intra-axial infection, which can lead to abscess if not treated.

Ventriculitis: Usually the result of a shunt placement, or intrathecal chemo. The ventricle will enhance and you can sometimes see ventricular fluid-fluid levels. If septa start to develop you can end up with obstructive patterns of hydrocephalus. The intraventricular extension of abscess is a very serious / ominous “pre-terminal event”.

Brain Tumors

My idea for the section on brain tumors is to provide a systematic approach, followed with some trivia that is likely to be asked. Remember when you are reading to continuously ask yourself - how can this information be written as a question? How would I write this question? Don't think like a doctor, think like some dummkopf trying to write multiple choice questions.

The first thing to do is to ask yourself is this a tumor, or is it a mimic? Mimics would be abscess, infarct, or a big MS plaque. This can be tricky.

My strategy is to go in this order:

(1) Patient age -> (2) Location of Mass -> (3) Characteristics.

People get hung up on characteristics and it confuses them. Only use that once your DDx is narrow already. Peds vs Adult is going to really cut up the possibilities. Then location is going to push it even further. Once you've got past location and age - than it's usually a "This vs That" with signal characteristics.

Age:

Peds		Adults	
Supratentorial	Infratentorial	Supratentorial	Infratentorial
Astrocytoma (including GBM)	JPA	iVJLCla ^ i	JPA
PXA (Pleomorphic Xanthoastrocytoma)	Medulloblastoma	Astrocytoma (including GBM)	Hemangioblastoma
PNET	Ependymoma	Oligodendroglioma	
DNET	Brainstem		
	Astrocytoma		
Ganglioglioma			

Location

The Brant and Helms discussion on brain tumors will have you asking “intra-axial” vs “extra-axial” first. This is not always that simple, but it does lend itself very well to multiple choice test questions (therefore it’s high yield).

Basically you need to memorize the “signs of extra-axial location”

<ul style="list-style-type: none"> • CSF Cleft • Displaced Subarachnoid Vessels • Cortical Gray matter between the mass and white matter • Displaced and expanded Subarachnoid spaces • Broad Dural Base / Tail • Bony Reaction 	<p>Sneaky Trick</p> <p>Usually Bony Reaction = Extra Axial</p> <p>BUT, Intra axial tumors like DNET and Ganglioglioma can focally thin the skull.</p>
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With that out of the way, I look at location as either, supratentorial, infratentorial, or one of the following 5 locations (skull base, sella, intraventricular, CP Angle, Pineal)

Skull Base	Sella / Parasellar (SATCHMO)	Intraventricular	CP Angle	Pineal Region
Chordoma (<i>midline</i>)	Sarcoid	Ependymoma	Schwannoma	Pineocytoma
Chondrosarcoma	Aneurysm, Adenoma	Subependymoma	Meningioma	Pineoblastoma
(<i>off midline</i>)	Rathke’s Cyst	Choroid Plexus Papilloma	Epidermoid	PNET
Esthesioneuroblastoma	Teratoma	Central	Arachnoid Cyst	Tectal Glioma
Sinonasal Carcinoma	Craniopharyngioma	Neurocytoma		Meningioma
Mets	Hamartoma	Colloid Cyst		Dermoid
Lymphoma	Hypothalamic Glioma	Meningioma		Germinoma
Paraganglioma	Meningioma	Giant Cell Astrocytoma		
	Optic Nerve Glioma			

Another location to consider is “Cortically Based”. Most intra-axial tumors are located in the white matter. So when a tumor spreads to or is primarily located in the gray matter you get a shorter DDx.

Cortically Based (DOG):

- Dysembryoplastic Neuroepithelial Tumor (DNET)
- Oligodendroglioma,
- Ganglioglioma

Another high yield piece of trivia regarding the cortical tumor / cortical met is that they often have *very little edema* and so a *small cortical met can be occult without IVcontrast*.

Signal Characteristics:

“*Multifocal Disease*”- Multiple tumors usually means mets (although 50% of mets are solitary , so don’t be fooled). The alternative to mets is either a primary brain tumor that likes to be multifocal or a syndromic situation.

- *Tumors that like to be multifocal:* Lymphoma, Multicentric GBM, Gliomatosis Cerebri
- *Tumors that are multifocal from seeding:* Medulloblastoma, Ependymoma, GBM, Oligodendroglioma
- Syndromes:

NF1	NF 2 “MSME”	Tuberous Sclerosis	VHL
Optic Gliomas	Multiple Schwannomas	Subependymal Tubers	Hemangioblastomas
Astrocytomas	Meningiomas	IV Giant Cell Astrocytomas	
	Ependymomas		

“Enhancement Why do things enhance? Or it might be better to ask , why “don’t” things enhance ? Understanding this makes it so much easier to remember enhancement. Things “don’t enhance” because of the intact blood brain barrier. So why do things enhance? Things enhance when they are either (1) outside the blood brain barrier - like an extra-axial tumor, or (2) they are high enough grade to disrupt the barrier - like a GBM. So all extra-axial tumors enhance (meningioma, schwannomas, pineal region, pituitary region), and all high grade tumors enhance (GBM..etc..). The exceptions to the rule are gangliogliomas and pilocytic astrocytoma (JPA) which are low grade tumors that enhance. **Obviously; exceptions make great test questions.*

No enhancement is seen in low grade astrocytomas (not counting JPAs), or cystic non-tumoral lesions (Dermoids, Epidermoids, Arachnoid Cyst).

“Ring Enhancement” - The mnemonic I like is the MAGIC DR one.

- Mets
- Abscess
- GBM
- Infarct (subacute phase)
- Contusion
- Demylinating (*open ring*)
- Radiation Necrosis / Resolving Hematoma

“Restriction” - If they show a supratentorial case with restriction it’s likely to be one of two things (1) **Abscess** or (2) **Lymphoma**. Technically any hypercellular tumor can restrict (GBM), but lymphoma is the one they classically show restricting. If it’s a CP angle case, then it’s an **epidermoid**. Lastly, a dirty move could be to show **Herpes** encephalitis restricting in the temporal horns.

“Midline Crossing” If they show it crossing the midline its most likely going to be a GBM **or Lymphoma**/ Alternatively sneaky things they could show doing this would be **radiation necrosis**, a big MS plaque in the corpus callosum, or Meningioma of the falx simulating a midline cross.

“Calcification” If they show it in the brain it is probably an **Oligodendroglioma**. The trick is that Oligodendrogliomas calcify 90% of the time by CT (and 100% by histopathology), whereas astrocytoma only calcify 20% of the time. But astrocytoma is very common and oligodendroglioma is not. So in other words in real life it’s probably still an astrocytoma.

“77 Bright” Most tumors are T1 dark (or intermediate). Exceptions might include a tumor that has bled (Pituitary apoplexy, or hemorrhagic mets). Hemorrhagic mets are classically seen on MR and CT (Melanoma, Renal, Carcinoid / Choriocarcinoma , Thyroid). Tumors with fat will also be T1 bright (Lipoma, Dermoid). Melanin is T1 bright (Melanoma). Lastly think about cholesterol in a colloid cyst.

T1 Bright:

Fat: Dermoid, Lipoma

Melanin: Melanoma

Blood: Bleeding Met or Tumor

Cholesterol: Colloid Cyst

“The Usual Characters”

Adult - Supratentorial

Astrocytoma: Most common primary brain tumor in adults. Tumors fitting in the category include Pilocytic Astrocytoma (WHO1), Diffuse Astrocytoma (WHO 2), Anaplastic Astrocytoma (WHO 3) , and GBM (WHO 4). Remember that low grade tumors don’t typically enhance (WHO2) and higher grades do (GBM and some Anaplastics). The exception to this rule is the pilocytic astrocytoma which often has an enhancing nodule. GBM is the beast that cannot be stopped. It grows rapidly, it necrosis, it crosses the midline, and it can restrict on diffusion. Remember **Turcot Syndrome** (*that GI polyp thing*) is associated with GBMs.

Gliomatosis Cerebri: A diffuse glioma with extensive infiltration. It involves at least 3 lobes and is often bilateral. The finding is usually mild blurring of the gray-white differentiation of CT, with extensive T2 hyperintensity and little mass effect on MR. It’s low grade, so it **doesn’t typically enhance**.

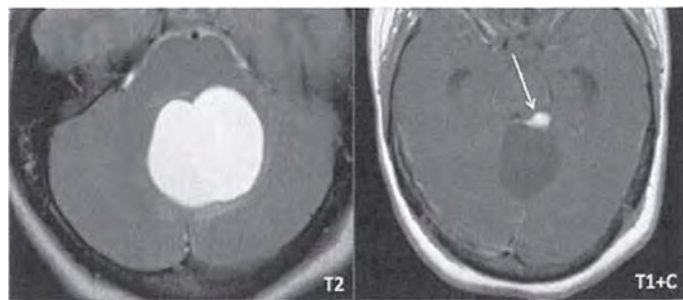
Oligodendroglioma: Remember this is the guy that **calcifies 90% of the time**. It’s most common in the frontal lobe and the buzzword is “**expands the cortex**”. This takes after its most specific feature of cortical infiltration and marked thickening. It’s likely you could get asked about this **1p/19q deletion** which apparently has a better outcome.

Primary CNS Lymphoma: Seen in end stage AIDS patients, and those post-transplant. EB virus plays a role. Most common type in **non-hodgkin B cell**. *Classic picture would be an intensely enhancing homogeneous solid mass in the periventricular region, with restricted diffusion*. However, it can literally look like and do anything. Classic Multiple choice test question is that it is **Thallium Positive on Spect** (toxo is not).

Metastatic Tumors: The most common CNS neoplasm. Usually from Lung or Breast. More than 80% are located at the gray-white junction (secondary to vessel caliber change in this region). Usually mets have more surrounding edema when compared to primary neoplasms of similar size. Remember that melanoma will be T1 bright (this is a common test question). Remember the MRCT mnemonic for bleeding mets (Melanoma, Renal, Carcinoid / Choriocarcinoma, Thyroid).

Adult - Infratentorial

Hemangioblastoma: First things first - immediately think about this when you see **cyst with a nodule** in an adult. Then think **Von Hippel Lindau**, especially if they are multiple. Often these things cause hydrocephalus.



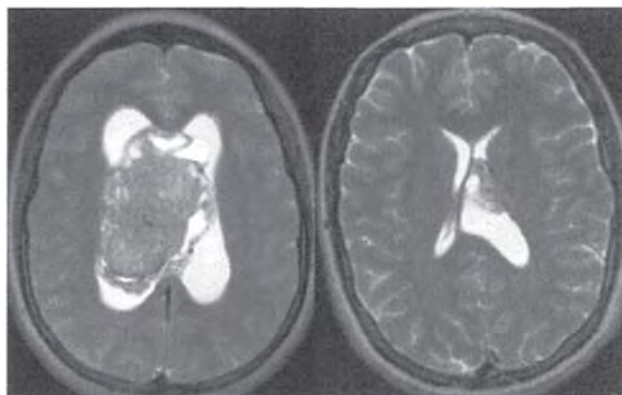
Hemangioblastoma - Cyst + Enhancing Nodule in Adult

Adult - Intraventricular

Subependymoma: Well circumscribed IV masses **most commonly at the foramen of Monro and the 4th ventricle**. They can cause hydrocephalus. They typically don't enhance. They are T2 bright (like most tumors).

Central Neurocytoma: This is the *most common IV mass in an adult aged 20-40*.

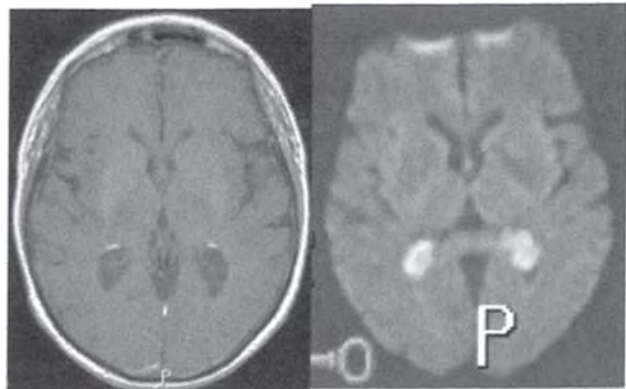
The buzzword is “**swiss cheese**,” because of the numerous cystic spaces on T2. They **calcify** a lot (almost like oligodendrogliomas).



Central Neurocytoma - Two examples, Cystic IV Mass

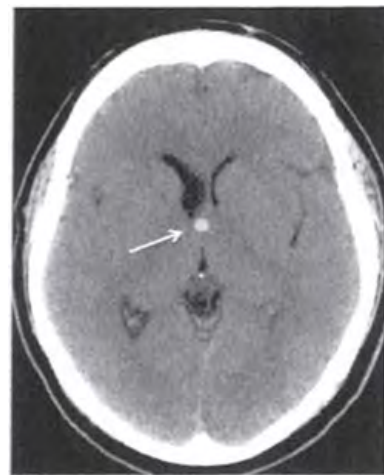
Xanthogranuloma - This is a benign choroid plexus mass. You see it all the time (7%) and don't even notice it.

The trick is that they restrict on diffusion, so they are trying to trick you into working them up. They are benign... leave them alone.



Xanthogranuloma - *Note the Restricted Diffusion*

Colloid Cyst - These are found almost exclusively in the anterior part of the 3rd ventricle behind the foramen of Monro. They **can cause sudden death via acute onset hydrocephalus**. Their appearance is somewhat variable and depends on what they are made of. If they have cholesterol they will be T1 bright, T2 dark. If they don't, they can be T2 bright. The trick is a round well circumscribed mass in the anterior 3rd ventricle. If shown on CT, it will be pretty dense.



Colloid Cyst -
- *Anterior 3rd Ventricle*
- *Hyperdense on CT*

Meningioma - *Can occur in an intraventricular location, most commonly (80%) at the trigone of the lateral ventricles (slightly more on the left). Details are discussed below.*

Adult - Sellar / Suprasellar

Pituitary Adenoma - The most common tumor of the sella. They are seen 97% of the time in adults. If they are greater than 1cm they are "macroadenomas." When functional, most are prolactin secreting (especially in women). Symptoms are easy to pick up in women (menstrual irregularity, galactorrhea). Men tend to present later because their symptoms are more vague (decreased libido). On MR, 80% are T1 dark and T2 bright. They take up contrast more slowly than normal brain parenchyma.

Things to know (about Pituitary adenomas):

- Microadenoma under 10mm,
- Macroadenoma over 10mm.
- Microadenomas typically form in the adenohypophysis (front 2/3).
- Prolactinoma is the most common functional type.
- Typically they enhance less than normal pituitary.

Pituitary' Anatomy Refresher

FLAT - PEG

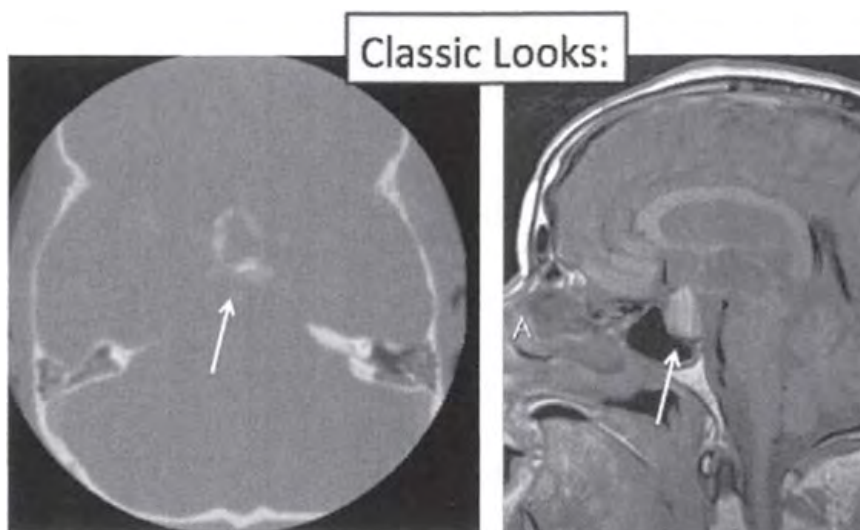
FLAT is in the front
-FSH, LSH, ADH, TSH

PEG is back
-Prolactin, Endorphins, GSH

Pituitary Apoplexy - Hemorrhage or Infarction of the pituitary, usually into an enlarged gland (either from pregnancy or a macroadenoma). Here are the multiple choice trivia association: taking **bromocriptine** (or other prolactin drugs), “**Sheehan Syndrome**” in pregnant woman, Cerebral Angiography. They will be **T1 bright** (remember adenoma is usually **T1 dark**). Supposedly this is an emergent finding because the lack of hormones can cause hypotension.

Craniopharyngioma - They come in two flavors: (a) Papillary and (b) Adamantinomatous. The Papillary type is the adult type (Papi for Pappi). They are solid and do not have calcifications. They recur less frequently than the Adamantinomatous form (because they are encapsulated). They strongly enhance. The relationship to the optic chiasm is key for surgery. Pediatric type is discussed below (under peds tumors).

Rathke Cleft Cyst - Usually an incidental finding, that is rarely symptomatic. They are variable on T1 and T2, but are usually very bright on T2. They do NOT enhance.



Craniopharyngioma
-Shown on bone window
-Calcifications in the Sella

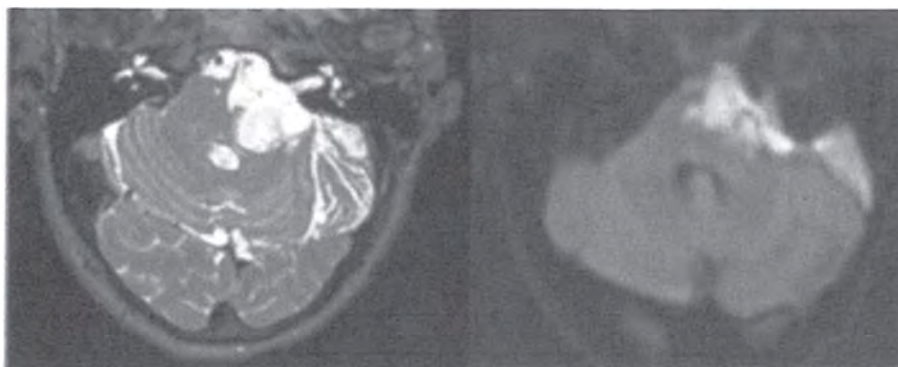
Pituitary Apoplexy
-Shown on T1
-T1 Bright Pituitary

Meningioma - Very Common extradural mass. One of the few brain tumors that is more common in women. They can calcify, and if you are lucky they will have a dural tail (which is pretty close to pathognomonic - with a few rare exceptions). Because they are extradural they will enhance strongly. Radiation of the head is known to cause meningiomas. The most common location (if anyone would ask) is over the cerebral convexity. They take up octreotide and Tc-MDP on Nuclear Medicine tests (a sneaky way to show this).

Vestibular Schwannoma - These guys account for 75% of CPA masses. When they are bilateral you should immediately think NF-2 (*one for each side*). Enhances strongly but more heterogeneous than meningiomas. May widen the porus acousticus resulting in a “trumpet shaped” IAC.

Meningioma	Schwannoma
Enhance Homogeneously	Enhance Less Homogeneously
Don't Usually Invade IAC	Invade IAC
Calcify more often	IAC can have “trumpeted” appearance

Epidermoid - Can be congenital or acquired (after trauma - classically after LP in the spine). Unlike dermoids they are usually off midline. They will follow CSF density and intensity on CT and MRI (*the exception is this zebra called a “white epidermoid” which is T1 bright -just forget I ever mentioned it*). The key points are (1) unlike an arachnoid cyst they are bright on FLAIR (sometimes warm - *they don't completely null*), and (2) they will restrict with diffusion.



Epidermoid : *Follows CSF Signal- Restricts Diffusion*

Arachnoid Cyst - Common benign lesions that is located within the subarachnoid space and contains CSF. They are increased in frequency in mucopolysaccharidoses (as are perivascular spaces). They are **dark on FLAIR** (like CSF), and **will NOT restrict with diffusion**.

How can you tell an epidermoid from an arachnoid cyst?

The epidermoid restricts, the arachnoid cyst does NOT.

Dermoid Cyst- Usually midline, and usually are found in 3rd decade. They contain lipoid material and are usually hypodense on CT and very bright on T1. They are associated with NF2.

Adult - Meningeal

Meningioma - As described above it is common and enhances homogeneously. The most common location is over the cerebral convexity and it has been known to cause hyperostosis.

Hemangiopericytoma - This is a soft tissue sarcoma that can **mimic an aggressive meningioma**, because they both enhance homogeneously. They also can mimic a dural tail, with a narrow base of dural attachment. They **won't calcify or cause hyperostosis , hut will invade the skull**.

Mets - The most common met to the dura is from breast cancer. 80% will be at the gray-white junction. They will have more edema than a primary tumor of similar size. Metastatic melanoma will be intrinsically T1 bright.

Peds Brain Tumors:

With regard to brain tumors occurring in kids, it can be useful to think about them as (a) tumors occurring in the first year of life (b) tumors of early adult hood - in addition to location.

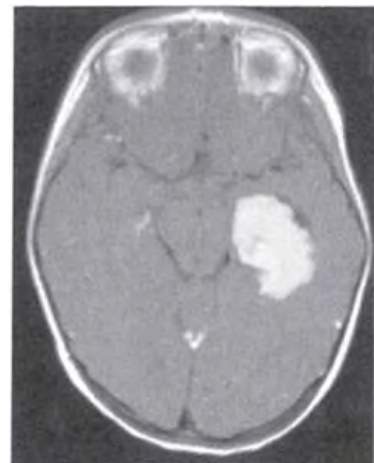
Tumors in the First Year Of Life:

Atypical Teratoma / Rhabdoid - These are highly malignant tumors (WHO IV), and rarely occur in patient's older than 6 years. The average age is actually 2 years, but they certainly occur in the first year of life. They can occur in supra and infratentorial locations (most common in the cerebellum). These are usually **large, pissed off looking tumors with necrosis** and heterogeneous enhancement.

Desmoplastic Infantile Ganglioglioma /Astrocytoma "DIG": These guys are **large cystic tumors** that like to involve the superficial cerebral cortex and leptomeninges. Unlike the Atypical Teratoma / Rhabdoid, these have an ok prognosis. They **ALWAYS arise in the supratentorial location** usually involving more than one lobe (frontal and parietal most commonly), and usually present before the first birthday. **Buzzword is "rapidly increasing head circumference."**

Choroid Plexus Papilloma / Carcinoma: Can occur in peds (85% under the age of 5) or adults. They make up about 15% of brain tumors in kids under one. Basically you are dealing with an intraventricular mass, which is often making CSF, so it causes hydrocephalus. *Here is the trick, brain tumors are usually supratentorial in adults and posterior fossa in kids. This tumor is an exception.*

In Adults iPs in the 4th Ventricle, in kids it's in the lateral ventricle (usually trigone).



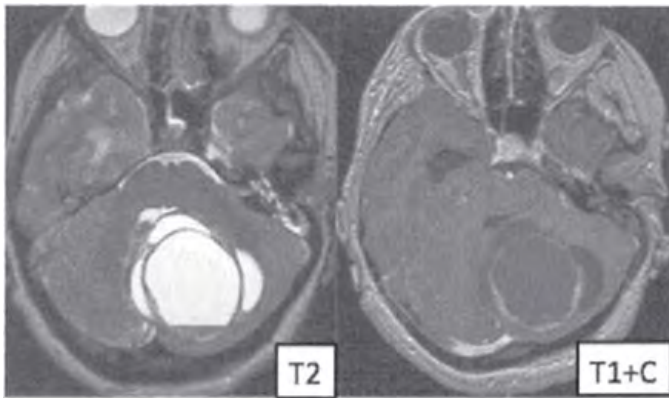
Choroid Plexus Papilloma
Avidly Enhancing in the Lateral Ventricle

Other trivia; it calcifies 25% of the time, angiography may show enlarged choroidal arteries which shunt blood to the tumor, the carcinoma type of this tumor looks very similar (unless it's invading the parenchyma) and is almost exclusively seen in kids.

Mets: In a child this will mean **neuroblastoma**. Key points to know about neuroblastoma mets is that they **like the bones and like the dura**. Involvement of the brain parenchyma or leptomeninges can happen but is rare. Also the classic move is to show coronal MRI or CT through the orbits as **neuroblastoma characteristically involves the posterolateral part of the orbit** where the frontal bone and greater wing of the sphenoid meet (EG can do this too).

Early Adult Tumors:

Juvenile Pilocytic Astrocytoma: Just think cyst with a nodule in a kid. They are WHO grade 1, but the nodule will still enhance. This will be located in the posterior fossa (or optic chiasm).



Pilocytic Astrocytoma : *Cyst + Nodule in Kid*

Brain Stem Glioma: Most common location is the pons, which is usually a high grade fibrillary glioma. It's going to be T2 bright with subtle or no enhancement. The imaging features are so classic that no biopsy is needed.

Ganglioglioma: This guy can occur at any age, anywhere, and look like anything. However, for the purpose of multiple choice testing the classic scenario would be a 13 year old with seizures, and a temporal lobe mass that is cystic and solid with focal calcifications. There may be overlying bony remodeling.

DNET (Dysembryoplastic Neuroepithelial Tumor): Kid with **drug resistant seizures**. The mass will always be in the **temporal lobe** (on the test - real life 60% temporal). Focal cortical dysplasia is seen in 80% of the cases. It is hypodense on CT, and on MRI there will be little if any surrounding edema. High T2 signal **“bubbly lesion.”**

PXA (Pleomorphic Xanthroastrocytoma): Superficial tumor that is ALWAYS supratentorial and usually involves the **temporal lobe**. They are often in the **cyst with a nodule** category (50%). There is usually no peritumoral T2 signal. The tumor frequently invades the leptomeninges. Looks just like a Desmoplastic Infantile Ganglioglioma - but is not in an infant.

Subependymal Giant Cell Astrocytoma: This is going to be shown in the setting of TS. They will show you renal AMLs or tell you the kid has seizures / developmental delay. It will arise from the wall of the lateral wall of the ventricle, often causing hydrocephalus. It enhances homogeneously.

PNET(Primitive Neuroectodermal Tumor): Histologically the same as a medulloblastoma. This one is supratentorial (deep white matter). It's classically very heterogeneous and known for metting outside the CNS.

Craniopharyngioma - As stated above, they come in two flavors: (a) Papillary and (b) Adamantinomatous. The kid type is the Adamantinomatous form. These guys are **calcified** (papillary is not). These guys recur more (Papillary does less - because it has a capsule). Buzzword is "machinery oil."

Hypothalamic Hamartoma - A classic Aunt Minnie. This is a hamartoma of the tuber cinereum (part of the hypothalamus located between the mammillary bodies and the optic chiasm). They are T1 and T2 iso and they do NOT enhance.

The classic history is **gelastic seizures** (although precocious puberty is actually more common).



Hamartoma of the
Tuber Cinereum

Medulloblastoma: These guys are cerebellar (arise from vermis - project into 4th ventricle) tumors that love to met via CSF pathways. The mass is heterogeneous on T1 and T2, and enhances homogeneously. They are much more common than their chief differential consideration the Ependymoma. They are hypercellular and may restrict. They calcify 20% of the time (less than Ependymoma). As mentioned above, they like to “drop met.” The buzzword is “*zuckerguss*” which apparently is German for sugar icing, as seen on post contrast imaging of the brain and spinal cord. As a point of absolute trivia, they are associated with *Basal Cell Nevus Syndrome*, and *Turcots Syndrome*.



Gorlin Syndrome - If you see a **medulloblastoma** next look for **dural calcs**. If you see thick dural calcs you might be dealing with this syndrome. They get **basal cell** skin cancer after radiation, and have odontogenic cysts.

Ependymoma: Bimodal distribution on this one (peak 1 around 6 years of age, peak 2 around 40 years old). The most common location is the floor of the 4th ventricle, with frequent extension into the foramen of Luschka and Magendie. They are the so called “plastic tumor” or “tooth paste” tumor because they squeeze out of the 4th ventricle. They enhance heterogeneously.

Medulloblastoma	Ependymoma
More common	Less Common
Originate from Vermis	
Can project into 4 th ventricle, do NOT usually extend into basal cisterns	Can extend into basal cisterns like tooth paste pushing through foramina of Luschka and Magendie
Enhance Homogeneously (<i>more so than Ependymoma anyway</i>)	Enhance Heterogeneously
Calcify Less (20%)	Calcify More (50%)
Linear “icing-like” enhancement of the brain surface is referred to as “Zuckerguss”	

Pineal Region Tumors:

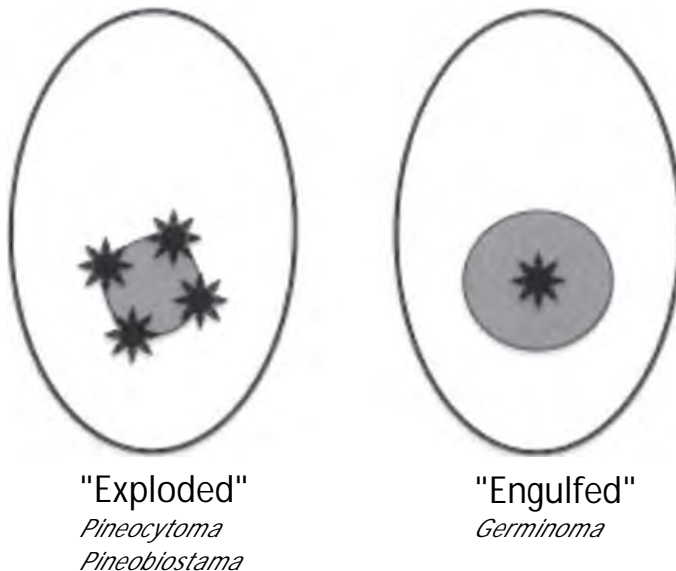
There are 3 main characters here, all of which can present with “vertical gaze palsy” (dorsal Parinaud syndrome).

Germinoma: The **more common of the 3**, and seen almost exclusively in boys (Germinomas in the suprasellar region are usually in girls). Precocious puberty may occur from secretion of hCG. Characteristic findings are a **mass containing fat and calcification** with variable contrast enhancement. It is heterogeneous on T1 and T2 (because of its mixed components).

Pineocytoma: Rare in childhood. Well-circumscribed, and **non-invasive**. Tend to be more solid, and the solid components do typically enhance.

Pineoblastoma: Does occur in childhood. Unlike the pineocytoma these guys are **highly invasive**. Some people like to think of these as PNETs in the pineal gland. They are **associated with retinoblastoma**. They are heterogeneous and enhance vividly.

Calcification Patterns in Pineal Tumors



Syndromes:

NF-1	Optic Nerve Gliomas
NF-2	MSME: Multiple Schwannomas, Meningiomas, Ependymomas
VHL	Hemangioblastoma (brain and retina)
TS	Subependymal Giant Cell Astrocytoma, Cortical Tubers
Nevoid Basal Cell Syndrome (Gorlin)	Medulloblastoma
Turcot	GBM, Medulloblastoma
Cowdens	Lhermitte-Duclos (Dysplastic cerebellar gangliocytoma)

Trauma

Parenchymal Contusion: The rough part of the skull base can scrape the brain as it slides around in a high speed MVA. Typical locations include the anterior temporal lobes and inferior frontal lobes. The concept of coup (site of direct injury) and contre-coup (opposite side of brain along vector of force). Contusion can look like blood with associated edema in the expected regions.

Diffuse Axonal Injury/Shear Injury: There are multiple theories on why this happens (different density of white and gray matter etc...) they don't matter for practical purposes or for multiple choice.

Things to know:

- *Initial Head CT is often normal*
- *Favorite sites of DAI are the posterior corpus callosum, and GM- WM junction in the frontal and temporal lobes*
- *Multiple small T2 bright foci on MRI*

Subarachnoid Hemorrhage: Trauma is the most common cause. FLAIR is the most sensitive sequence. This is discussed in more detail below.

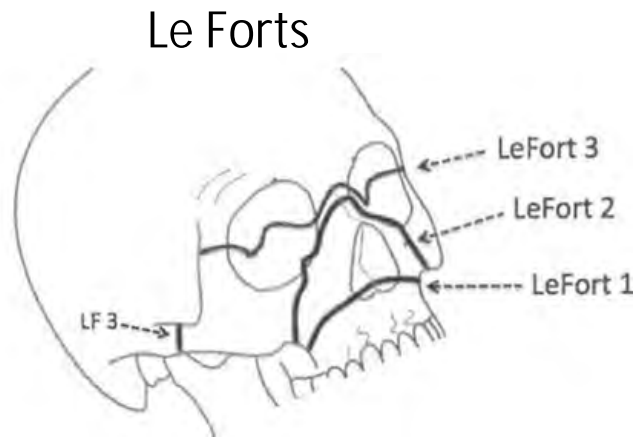
Subdural vs Epidural

Epidural	Subdural
Trauma Patient - with a skull fracture	Old man or alcoholic with an atrophic brain who likes to fall a lot, and stretch / tear those bridging veins.
“Bi-convex” or Lenticular	“Bi-concave”
Can cross the midline	Does not cross the midline, may extend into interhemispheric fissure
Can NOT cross a suture	Can cross a suture
Usually arterial	Usually venous
Can rapidly expand and kill you	

The LeFort Fracture Pattern System: In the dark ages, Rene LeFort beat the skull of cadavers with clubs and threw them off buildings. He then described three facial fracture patterns that interns in ENT and people who write multiple test questions think are important. It can be overly complicated but the most common way a test question is written about these is either by asking the buzzword, or the essential component.

Buzzwords:

- LeFort 1: “The Palate Separated from the Maxilla” or “Floating Palate”
- LeFort 2: “The Maxilla Separated from the Face or “Pyramidal”
- LeFort 3: “The Face Separated from the Cranium”



Essential Elements: All three fracture types share the pterygoid process fracture. If the pterygoid process is not involved, you don't have a LeFort. Each has a unique feature (which lends itself easily to multiple choice).

- LeFort 1: Lateral Nasal Aperture
- LeFort 2: Inferior Orbital Rim and Orbital Floor
- LeFort 3: Zygomatic Arch and Lateral Orbital Rim/Wall

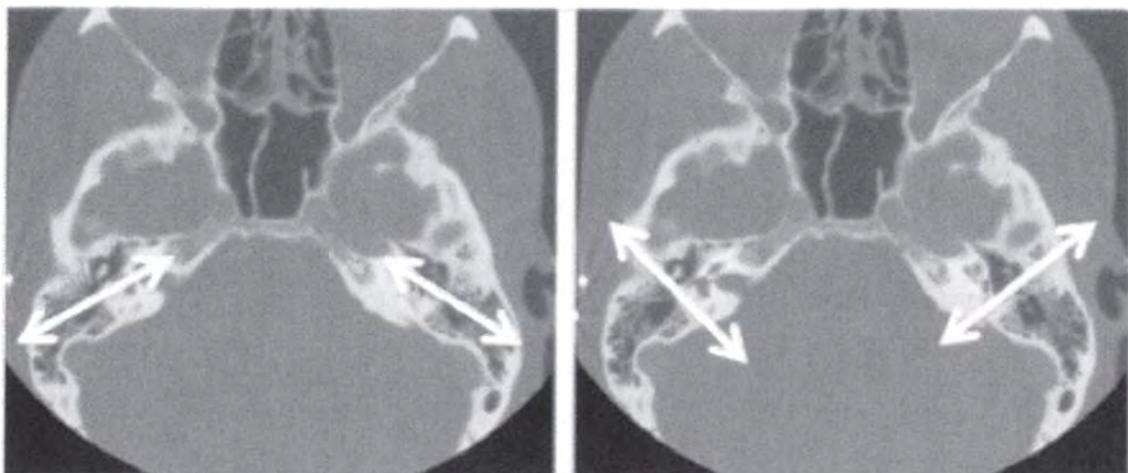
Things to Know About Facial Fractures:

- Nasal Bone is the most common fracture
- Zygomaticomaxillary Complex Fracture (Tripod) is the most common fracture pattern, and involves the zygoma, inferior orbit, and lateral orbit.
- Le-Fort Fractures are both a stupid and a high yield topic in facial trauma - for multiple choice. Floating Palate = 1, Pyramidal = 2, Separated Face = 3
- Transverse vs Longitudinal Temporal Bone Fractures - this classification system is stupid and outdated since most are mixed and otic capsule violation is a way better predictive factor... but this is still extremely high yield - see chart below.

Mucocele: If you have a fracture that disrupts the frontal sinus outflow tract (usually nasal-orbital-ethmoid types) you can develop adhesions, and obstruction of the sinus resulting in mucocele development. The **buzzword is “airless expanded sinus.”** They are usually T1 bright, with a thin rim of enhancement (tumors more often have solid enhancement). The frontal sinus is the most common location - occurring secondary to trauma (as described above).

Temporal Bone Fractures: The traditional way to classify these is longitudinal and transverse and this is almost certainly how the questions will be written. In the real world that system is old and worthless, as most fractures are complex with components of both. The real predictive finding of value is violation of the otic capsule - as described in more modern papers.

Longitudinal	Transverse
Long Axis of T-Bone	Short Axis of T-Bone
More Common	Less Common
More Ossicular Dislocation	More Vascular Injury (Carotid / Jugular)
Less Facial Nerve Damage (around 20%)	More Facial Nerve Damage (>30%)
More Conductive Hearing Loss	More Sensorineural Hearing Loss



Longitudinal

Transverse

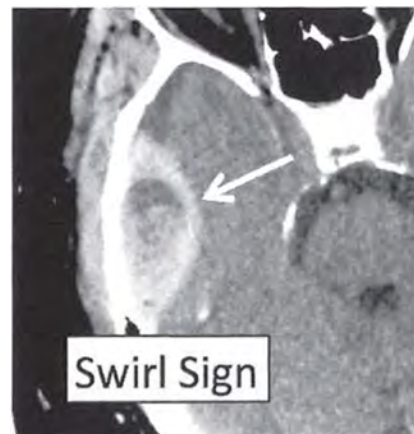
How old is that blood?

This is an extremely high yield topic. Maybe the most high yield topic in all of neuro, with regard to multiple choice. The question can be asked with CT or MRI (MRI more likely). If they do ask the question with CT it's most likely to be the subacute subdural that is isointense to brain, with loss sulci along the margins. They could also show the "swirl sign" - see below.

Blood on CT

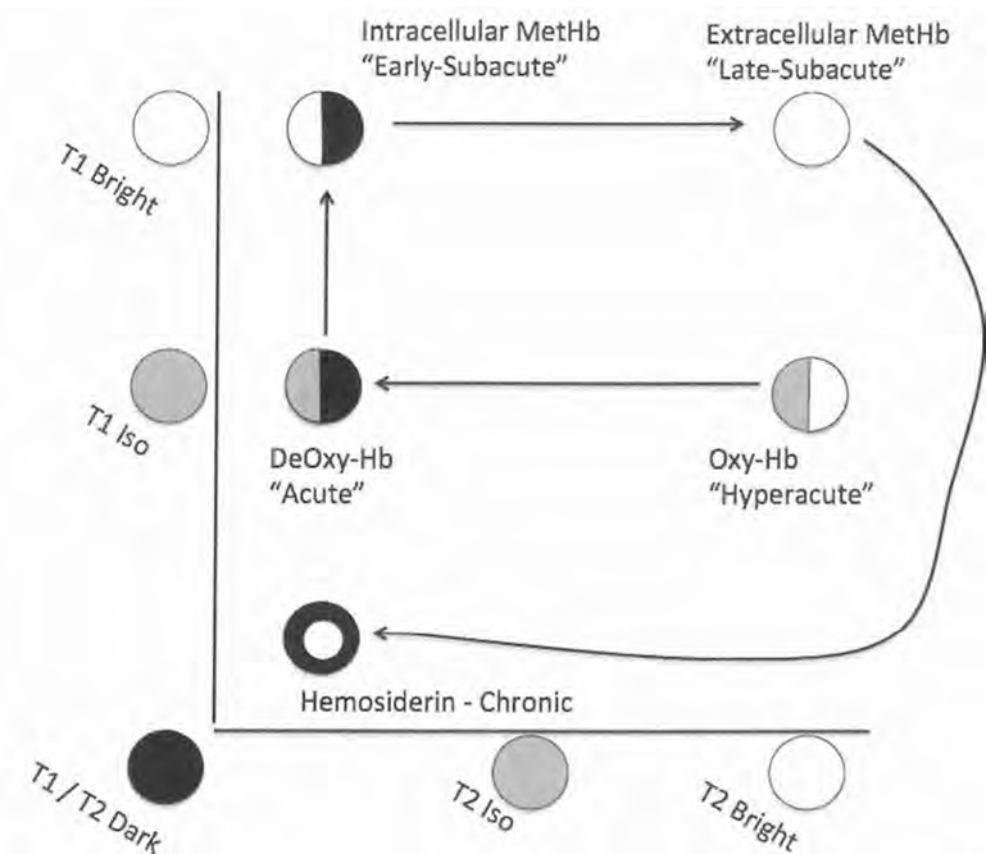
Hyperacute Acute (< 1 hour)	Hypodense
Acute (1 hour - 3 days)	Hyperdense
Subacute (4 days - 3 weeks)	Progressively less dense, eventually becoming isodense to brain. Peripheral rim enhancement may occur with contrast.
Chronic (> 3 weeks)	Hypodense

Swirl Sign - This is an ominous sign of active bleeding. The central low attenuation blood represents acute non-clotted blood, with surrounding more acute blood.



MRI is more difficult to remember. Some people use the mnemonic "IB, ID, BD, BB, DD" or "It Be Iddy Biddy, BaBy, Doo-Doo" which I find very irritating. I prefer mnemonics that employ known words (just my opinion). Another one with actual words is "George Washington Bridge" For T1 (Gray, White, Black), and Oreo Cookie for T2 (Black, White, Black).

Instead of memorizing baby babbling noises, I use this graph showing a clockwise movement.



Another strategy is to actually try and understand the MRI changes (I strongly discourage this).

Hyperacute	< 24 hours	Oxyhemoglobin, Intracellular	T1- Iso, T2 Bright
Acute	1-3 days	Deoxyhemoglobin, Intracellular	T1 - Iso, T2 Dark
Early Subacute	> 3 days	Methemoglobin, Intracellular	T1 Bright, T2 Dark
Late Subacute	>7 days	Methemoglobin, Extracellular	T1 Bright, T2 Bright
Chronic	> 14 days	Ferritin and Hemosiderin, Extracellular	T1/T2 Dark Peripherally, Center may be T2 bright

Hemorrhage (Non-Traumatic)

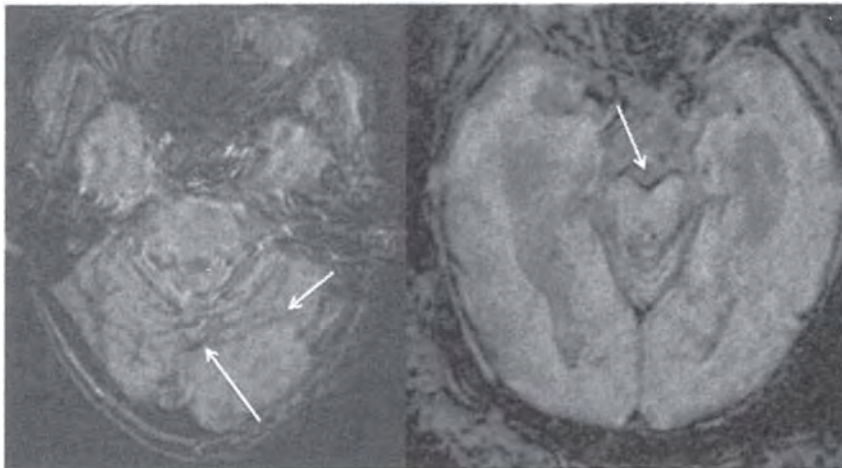
Subarachnoid Hemorrhage:

Yes, the most common cause is trauma. A common point of trivia is that the **most sensitive sequence on MRI for acute SAH is FLAIR** (because it won't suppress out - making it hyperintense). Be aware that **supplemental oxygen** (usually 50-100%) **can give you a fake out that looks like SAH on FLAIR**. When the blood is real, in the absence of trauma, there are a few other things to think about.

Sequella of SAH

- (1) Hydrocephalus - Early
- (2) Vasospasm - 7-10 days
- (3) Superficial Siderosis - Late

- **Aneurysm** - Discussed below.
- **Benign Non-Aneurysm Perimesencephalic hemorrhage:** This is a well described entity (although not well understood). This is NOT associated with aneurysm (usually - 95%), and may be associated with a venous bleed. The location of the blood - around the midbrain and pons without extension into the lateral Sylvian cisterns or interhemispheric fissures is classic. **Just think anterior to the brainstem.** Re-bleeding and ischemia are rare- and they do extremely well.
- **Superficial Siderosis:** This is a side effect of repeated episodes of SAH. I like to think about this as “*staining the surface of the brain with hemosiderin.*” The classic look is curvilinear **low signal on gradient coating the surface of the brain**. The classic history is **sensorineural hearing loss and ataxia**.



Superficial Siderosis - *Hemosiderin Staining*

Intraparenchymal Hemorrhage:

- **Hypertensive Hemorrhage:** Common locations are the basal ganglia, pons, and cerebellum. For the purpose of multiple choice tests the **basal ganglia is the most common location (specifically the putamen)**. You typically have intraventricular extension of blood.
- **Amyloid Angiopathy:** History of an old dialysis patient (or some other history to think Amyloid). *The classic look is multiple lobes at different ages with scattered microbleeds on gradient.*
- **Septic Emboli:** These are seen in certain clinical scenarios (**IV drug user**, organ transplant, cyanotic heart disease, AIDS patients, people with lung AVMs). **The classic look is numerous small foci of restricted diffusion. Septic emboli to the brain result in abscess, mycotic aneurysms (most commonly in the distal MCAs)**, The location favors the gray-white interface and the basal ganglia. There will be surrounding edema around the tiny abscesses. The classic scenario should be parenchymal bleed in a patient with infection.
- **Other random causes:** This would include AVMs, vasculitis, brain tumors (primary and mets) - these are discussed in greater detail in various section of the text.

Intraventricular Hemorrhage:

- Not as exciting. Just think about trauma, tumor, hypertension, AVMs, and aneurysms - all the usual players.

Epidural / Subdural Hemorrhage:

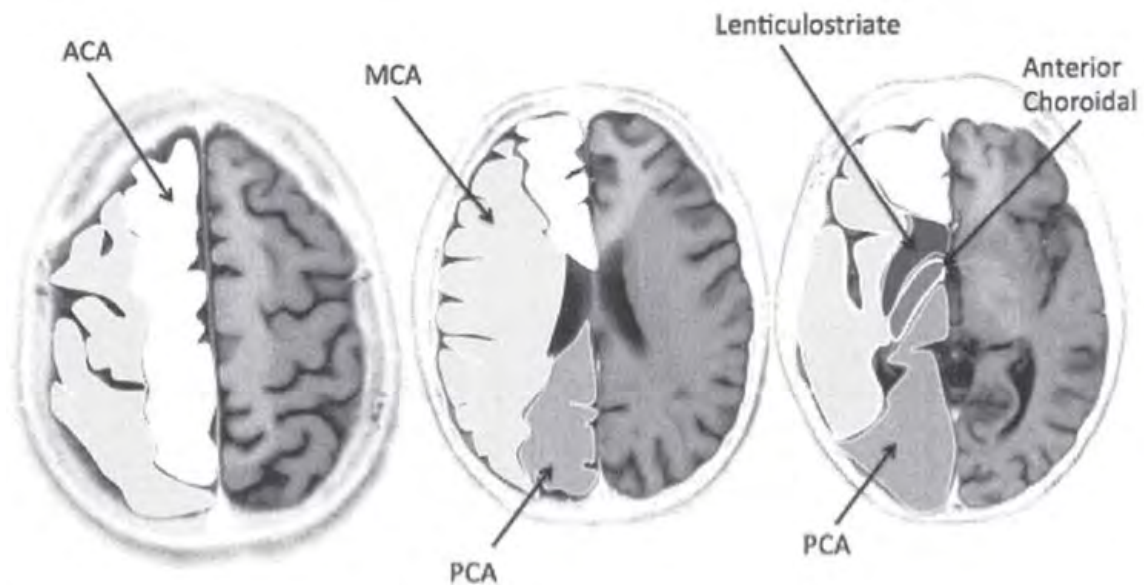
- Obviously these are usually post-traumatic.
- **Dural AVFs and High Flow AVMs** can bleed causing subdurals / subarachnoid spaces. These are discussed further, later in the chapter.

Vascular

Stroke -

Stroke is a high yield topic. You can broadly categorize stroke into ischemic (80%) and hemorrhagic (20%). It's critical to remember that stroke is a clinical diagnosis and that imaging findings compliment the diagnosis.

Watershed Zones: Below is a diagram showing the various vascular territories. The junction between these zones is sometimes referred to as a "Watershed". These areas are prone to ischemic injury, especially in the setting of hypotension or low oxygen states (near drowning or Roger Gracie's cross choke from the mount).



Gamesmanship: Watershed Infarcts in a Kid = Moyamoya

Imaging Signs on CT:

Dense MCA Sign	Intraluminal thrombus is dense, usually in the M1 and/or M2 segments
Insular Ribbon Sign	Loss of normal high density insular cortex from cytotoxic edema
Loss of GM-WM differentiation	Basal Ganglia / Internal Capsular Region and Subcortical regions
Mass Effect	Peaks to 3-5 days
Enhancement	Rule of 3s: Starts in 3 days, peaks in 3 weeks, gone by 3 months.

“Fogging” - This is a phase in the evolution of stroke when the infarcted brain looks like normal tissue. This is seen around 2-3 weeks post infarct. “Fogging” is classically described with non-contrast CT, but T2 MRI sequences have a similar effect (typically occurring around day 10). In the real world, you could give IV contrast to demarcate the area of infarct or just understand that fogging occurs.

Imaging Signs on MRI:

Restricted Diffusion: Acute infarcts usually are bright from about 30 mins after the stroke to about 2 weeks. **Restricted diffusion without bright signal on FLAIR should make you think hyperacute (< 6 hours).**

Things That Restrict Diffusion (Other than stroke)

Bacterial Abscess, CJD (cortical), Herpes, Epidermoids, Hypercellular Brain Tumors (Classic is lymphoma), Acute MS lesions, Oxyhemoglobin, and Post Ictal States. Also artifacts (susceptibility and T2 shine through).

Enhancement: The rule of 3's is still useful. Enhancement starts around day 3, it peaks around 3 weeks, and is gone by 3 months.

	0-6 hours	6-24 hours	24 hours -1 week
Diffusion	Bright	Bright	Bright
FLAIR	NOT BRIGHT	Bright	Bright
T1	Iso	Dark	Dark, with Bright Cortical Necrosis
T2	Iso	Bright	Bright

Hemorrhagic Transformation - This occurs in about 50% of infarcts, with the typical time period between 6 hours and 4 days. If you got TPA it's usually within 24 hours of treatment. People break these into (1) tiny specs in the gray matter called “petechial” which is the majority (90%) and (2) full on hematoma - about 10%.

Who gets it? People on anticoagulation, people who get TPA, people with embolic strokes (especially large ones), venous infarcts.

Predictors of Hemorrhagic Transformation in Patients Getting TPA

Multiple Strokes, Proximal MCA occlusion, **Greater than 1/3 of the MCA territory**, Greater than 6 hours since onset “delayed recanalization”, Absent collateral flow

Venous Infarct: Not all infarcts are arterial, you can also stroke secondary to venous occlusion (usually the sequelae of dural venous sinus thrombosis or deep cerebral vein thrombosis). In general, venous infarcts are at higher risk for hemorrhagic transformation. In little babies think dehydration, in older children think about mastoiditis, in adults think about coagulopathies (protein C & S def) and oral contraceptives. The most common site of thrombosis is the sagittal sinus, with associated infarct occurring 75% of the time.

Venous thrombosis can present as a dense sinus or “empty delta” on contrast. Venous infarcts tend to have *heterogeneous restricted diffusion*. Venous thrombosis can result in vasogenic edema that eventually progresses to stroke and cytotoxic edema.

- Arterial stroke = Cytotoxic Edema
- Venous Stroke = Vasogenic Edema + Cytotoxic Edema

Stigmata of chronic venous thrombosis include the development of a dural AVF, or increased CSF pressure from impaired drainage.

Aneurysm

Who gets them? People who smoke, polycystic kidney disease, connective tissue disorders (Marfans, Ehlers-Danlos), people with aortic coarctation, NF, FMD, and AVMs.

Where do they occur? They occur at branch points (why do persistent trigeminals get more aneurysms? - because they have more branch points). They favor the anterior circulation (90%) - with the **anterior communicating artery being the most common site**. As a piece of random trivia the basilar is the most common posterior circulation location (PICA origin is the second most common).

When do they rupture? Rupture risk is increased with size, a posterior location, history of prior SAH, smoking history, and female gender.

Which one did it? A common dilemma is SAH in the setting of multiple aneurysms. The things that can help you are location of the SAH/Clot, location of the vasospasm, size, and which one is the most irregular (*Focal out-pouching Murphy's tit*)

Aneurysm Types:

Saccular (Berry): The most common type. They are commonly seen at bifurcations. The underlying pathology may be a congenital deficiency of the internal elastic lamina and tunica media (at branch points). Remember that most are idiopathic (with the associations listed above). They are multiple 15-20% of the time.

Fusiform Aneurysm - Associated with PAN, Connective Tissue Disorders, or Syphilis. These more commonly affect the posterior circulation. May mimic a CPA mass.

Pseudo Aneurysm - Think about this with an irregular (often saccular) arterial out-pouching at a strange / atypical location. You may see focal hematoma next to the vessel on non-contrast.

- *Traumatic* - Often distal secondary to penetrating trauma or adjacent fracture.
- *Mycotic* - Often distal (most commonly in the MCA), with the associated history of endocarditis, meningitis, or thrombophlebitis.

Pedicle Aneurysm - Aneurysm associated with an AVM. The trivia to know is that it's **found on the artery feeding the AVM** (75% of the time). *These may be higher risk to bleed than the AVM itself (because they are high flow).*

Blister Aneurysm - This is a sneaky little dude (the angio is often negative). It's broad-based at a non-branch point (supraclinoid ICA is the most common site).

Infundibular Widening - Not a true aneurysm, but instead a funnel-shaped enlargement at the origin of the Posterior Communicating Artery at the junction with the ICA. *Thing to know is "not greater than 3mm."*

Saccular (Berry)	Branch Points - in the Anterior Circulation
Fusiform	Posterior Circulation
Pedicle Aneurysm	Artery feeding the AVM
Mycotic	Distal MCAs
Blister Aneurysm	Broad Based Non-Branch Point (Supraclinoid ICA)

Maximum Bleeding-Aneurysm Location

ACOM	Interhemispheric Fissure
PCOM	Ipsilateral Basal Cistern
MCA Trifurcation	Sylvian Fissure
Basilar Tip	Interpeduncular Cistern, or Intraventricular
PICA	Posterior Fossa or Intraventricular

Vascular Malformation:

There are 6 different kinds, and I will actually touch on all 6 - some in more detail than others.

- (1) **High Flow AVMs:** This is the most common type of high flow lesion. They favor a supratentorial location and are the result of a congenital malformation in the development of the capillary bed. As the name “high flow” implies there is an arterial component (arterial component -> nidus -> draining veins). Hemorrhage is the most common complication, and has a bleeding incidence of about 3% per year. Seizure would be the second most common complication, and the adjacent brain may be atrophic and gliotic.

* *Increased Bleeding Risk: Small size of A VM (actually higher pressure), single draining vein, intranidal/perinidal aneurysm, basal ganglia/thalamic/periventricular locations.*

- (2) **Dural AVF:** These can be high flow or low flow. They are less common than the high flow AVMs. Unlike the AVM these are thought to be acquired secondary to dural sinus thrombosis. Unlike AVMs there is no nidus. This presents in the 50s-60s (AVMs present in 20s-30s). **Classic symptom is pulsatile tinnitus, when it involves the sigmoid sinus.** May have vision problems if the cavernous sinus is involved.

* *Increased Bleeding Risk: Direct cortical venous drainage.*

* *Can be occult on MRI/MRA - need catheter angio if suspicion high*

- (3) **DVA:** This is not actually a vascular malformation, just a variation in normal venous drainage. If you tried to resect one, you would end up with a nice venous infarct. Some buzzwords are “caput medusa” or “large tree with multiple small branches” - to describe its appearance. The big thing to remember is the **association with cavernous malformations.**

- (4) **Cavernous Malformation:** These things are also called “cavernomas” and “cavernous angiomas.” These are low flow lesions with a dilated capillary bed **without intervening normal brain tissue.** They can be single or multiple (more common in Hispanics). Buzzword is “popcorn-like” with “peripheral rim of hemosiderin.” Look for them on gradient (because of the hemosiderin). They can ooze some blood, but typically don’t have full-on catastrophic bleeds. As mentioned above, they may have a nearby DVA.
- (5) **Capillary Telangiectasia:** This is another slow flow lesion, that unlike the cavernoma **does have intervening normal brain tissue.** They don’t bleed and are usually totally incidental. The most common look for this is a single lesion in the pons. Again these are best seen on gradient (slow flow and deoxyhemoglobin). The buzzword is “brush-like” or “stippled pattern” of enhancement. Key high yield fact is that **these can develop as a complication of radiation therapy.**
- (6) **Mixed** - This is a wastebasket term, most often used for DVA with AV shunting or DVAs with telangiectasias.

Vasospasm

Vessels do not like to be bathed in blood (SAH), it makes them freak out (spasm). The **classic timing for this is 4-14 days after SAH (NOT immediately).** It usually looks like smooth, long segments of stenosis. It typically involves multiple vascular territories. It can lead to stroke.

Who gets it? It’s usually for SAH and the more volume of SAH the greater the risk. In 1980 some neurosurgeon came up with this thing called the Fisher Score, which grades vasospasm risk. The gist of it is greater than 1mm in thickness or intraventricular / parenchymal extension is at higher risk.

Are there Non-SAH causes of vasospasm? Yep. Meningitis, PRES, and Migraine Headache.

Vascular Dissection

Vascular dissection can occur from a variety of etiologies (usually penetrating trauma, or a trip to the chiropractor). Penetrating trauma tends to favor the carotids, and blunt trauma tends to favor the vertebrae. This would be way too easy to show on CT as a flap, so if it’s shown it’s much more likely to be the T1 bright “crescent sign”, or intramural hematoma.

Vasculitis

You can have a variety of causes of CNS vasculitis. One way to think about it is by clumping it into (a) Primary CNS vasculitis, (b) Secondary CNS vasculitis from infection, or sarcoid, (c) systemic vasculitis with CNS involvement, and (d) CNS vasculitis from a systemic disease.

Primary CNS vasculitis	Primary Angiitis of the CNS (PACNS)
Secondary CNS vasculitis from infection, or sarcoid	Meningitis (bacterial, TB, Fungal). Septic Embolus, Sarcoid,
Systemic vasculitis with CNS involvement	PAN , Temporal Arteritis, Wegeners, Takayasu's,
CNS vasculitis from a Systemic Disease	Cocaine Use , RA, SLE, Lyme's

They all pretty much look the same with multiple segmental areas of vessel narrowing, with alternating dilation ("beaded appearance"). You can have focal areas of vascular occlusion.

Trivia:

- *PAN is the Most Common systemic vasculitis to involve the CNS (although it is a late finding).*
- *SLE is the Most Common Collagen Vascular Disease*

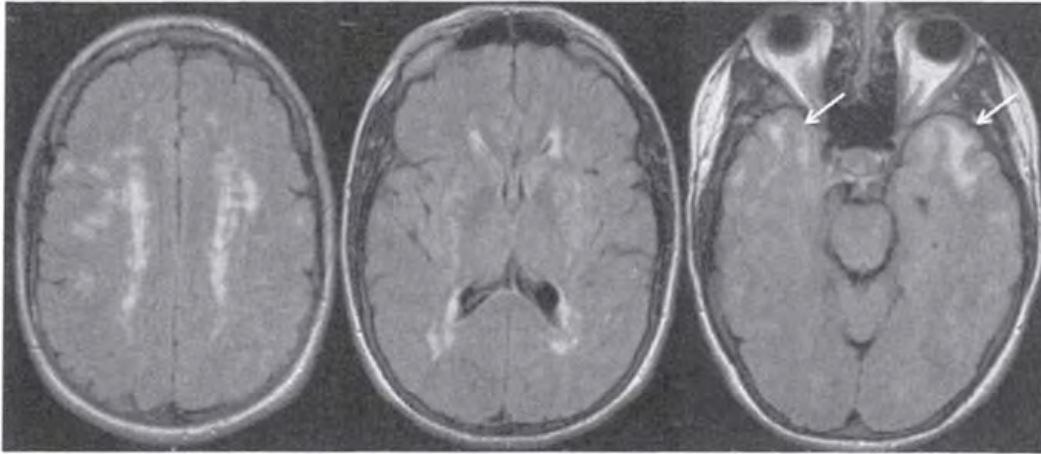
Misc Vascular Conditions

Moyamoya - This non-atherosclerotic poorly understood entity (originally described in Japan - hence the name), is characterized by progressive stenosis of the supraclinoid ICA eventually leading to occlusion. The progressive stenosis results in an enlargement of the basal perforating arteries.

Things to know:

- *Buzzword = "Puff of Smoke" - for angiographic appearance*
- *Watershed Distribution*
- *In a child think sickle cell*
- *Other notable associations include: NF, prior radiation, Downs syndrome*
- *Bi-Modal Age Distribution (early childhood, and middle age)*
- *Children Stroke, Adults Bleed*

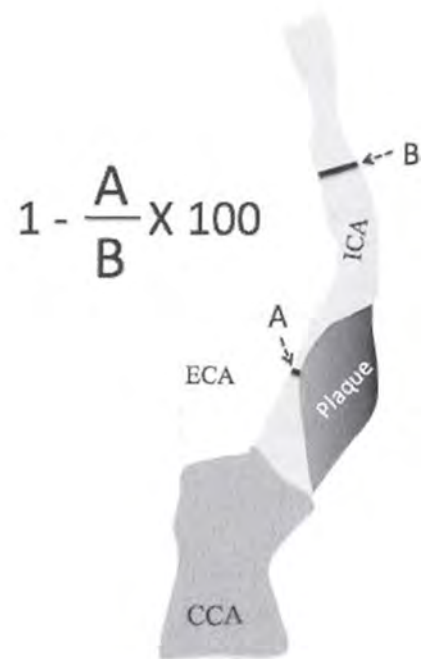
CADASIL - (Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy). Think about a **40 year old presenting with migraine headaches**, then eventually dementia. The MRI will show severe white matter disease involving multiple vascular territories, in the frontal and **temporal lobe**. The occipital lobes are often spared.



CADASiL - Diffuse White Matter Disease, Hitting the Temporals, Sparing Occipitals

NASCET Criteria: The North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria, is used for carotid stenosis. *The rule is: measure the degree of stenosis using the maximum internal carotid artery stenosis ("A ") compared to a parallel (non-curved) segment of the distal, cervical internal carotid artery ("B").*

You then use the formula $[1 - A/B] \times 100\%$.

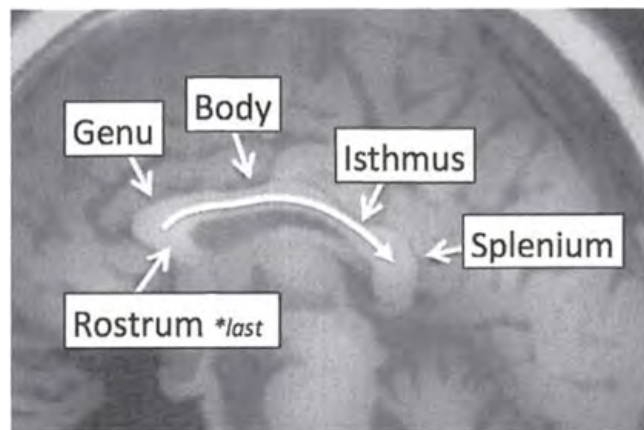


Developmental / Congenital

Congenital malformations is a very confusing and complicated topic, full of lots of long Latin and French sounding words. If we want to keep it simple and somewhat high yield you can look at it in 6 basic categories: (1) Failure to form, (2) Failure to cleave, (3) Failure to migrate, (4) Normal forming but massive insult makes you look like you didn't form (5) Herniation syndromes, and (6) Craniosynostosis.

Failure to Form:

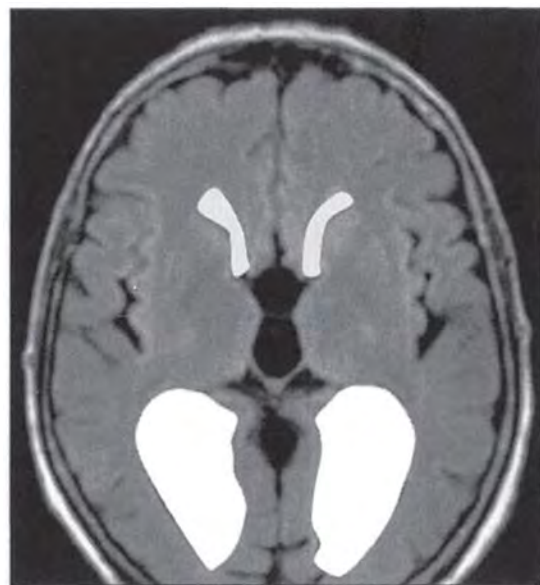
A classic point of trivia is that the corpus callosum forms front to back (then rostrum last). Therefore hypoplasia of the corpus callosum is usually absence of the splenium (with the genu intact).



With agenesis of the corpus callosum, a common trick is to show colpocephaly (asymmetric dilation of the occipital horns).

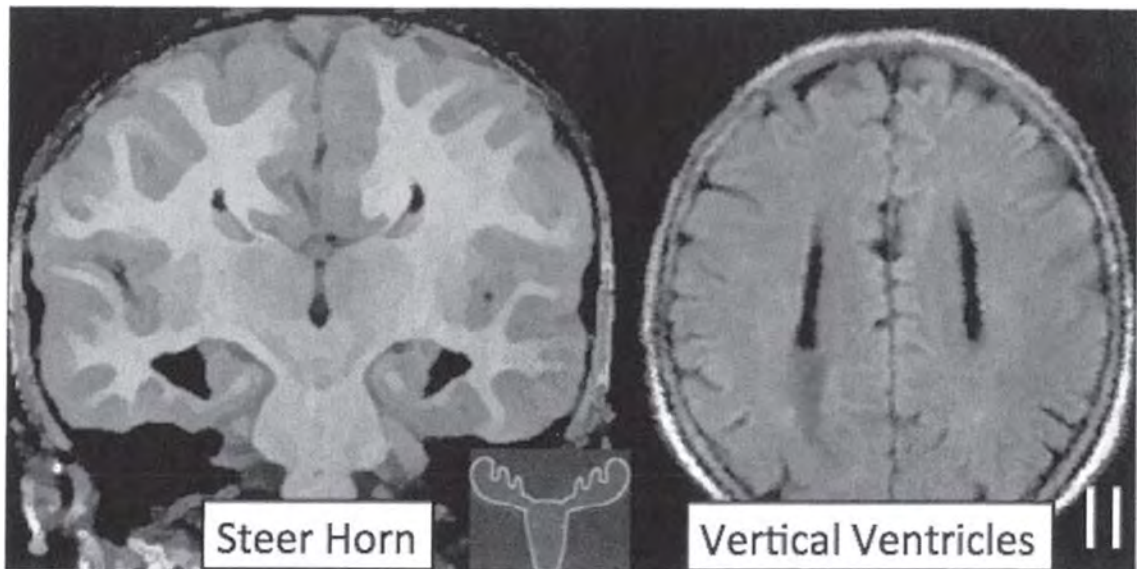
When you see this picture you should say two things:

- (1) Corpus Callosum Agenesis*
- (2) Pericallosal Lipoma*



Colpocephaly - Disproportionate prominence of the occipital horns

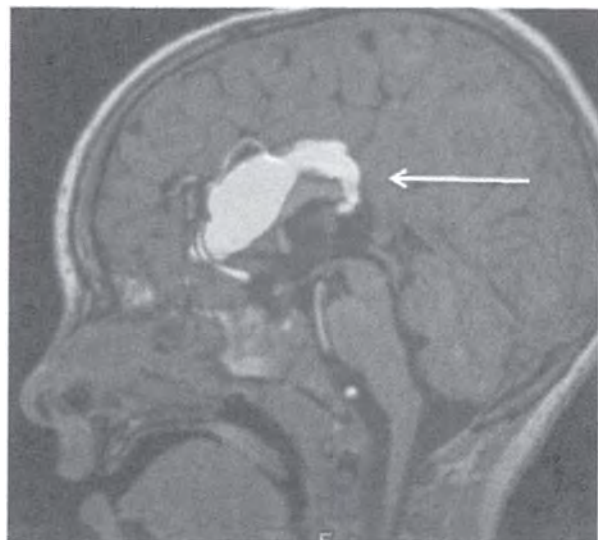
Callosal Dysgenesis / Agenesis: This is associated with lots of other syndromes/ malformations (Lipoma, heterotopias, schizencephaly, lissencephaly etc...). **“Most common anomaly seen with other CNS malformations.”** If they ask this, it will either be the colpocephaly picture, the **steer horn** appearance of coronal, or the **vertical ventricles** widely spaced (racing car) on axial.



Agenesis of the Corpus Callosum

Intracranial Lipoma: Associated with callosal dysgenesis. 50% are found in the interhemispheric fissure and if they show it that's where it will be.

Trivia: CNS Lipomas are congenital malformations, not true neoplasms. The tubulonodular type frequently calcify.



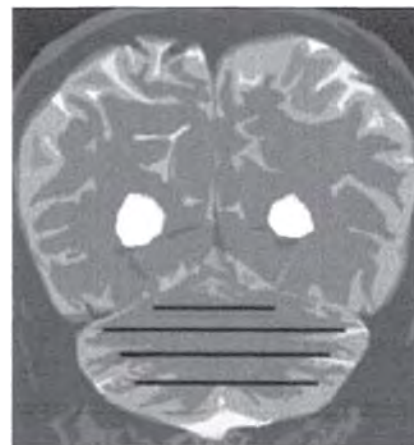
Intracranial Lipoma

Anencephaly: Neural tube fails to close on the cranial end leading to reduced or absent cerebrum and cerebellum. The hindbrain will be present. Obviously this is not compatible with life. Trivia: **AFP will be elevated, polyhydramnios** will be present (hard to swallow without a brain). If they show it, it will have to be antenatal ultrasound and will likely be polyhydramnios plus the incredibly creepy **“frog eye” appearance** on the coronal plane (due to absent cranial bone / brain with bulging orbits).

Iniiencephaly: Rare neural defect with the main features including deficit of the occipital bones. This results in an enlarged foramen magnum. These guys also have really jacked up spines. **“Star Gazing Fetus”** is the buzzword because they are contorted in a way that makes their face turn upward (hyper-extended cervical spine, short neck, and upturned face).

Arhinencephaly: No olfactory bulbs and tracts.
Seen with **Kallmann Syndrome** (hypogonadism, mental retardation).

Rhombencephalosynapsis: Congenital anomaly of the cerebellum, where the vermis doesn't develop and instead you just fuse your cerebellum. Classically a transversely oriented single lobed cerebellum is shown (this is an Aunt Minnie).



Rhombencephalosynapsis

Joubert Syndrome: OK so this is another Aunt Minnie, because of the **“Molar Tooth” appearance** of the superior cerebellar peduncles (they are elongated like the roots of a tooth). There is going to be a small or aplastic cerebellar vermis, and there will be absence of pyramidal decussation. It has a **strong association (50%) with retinal dysplasia**. There is also an association with multicystic dysplastic kidneys (30%). When you see it in combination with liver fibrosis it's a total zebra (near unicorn) called COACH syndrome.



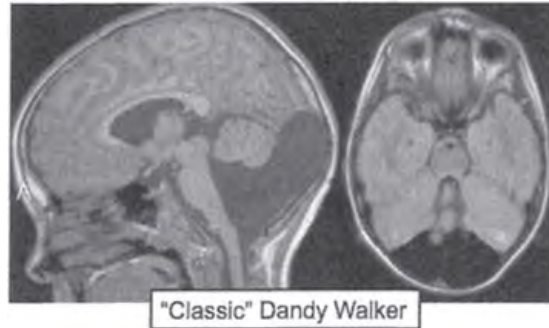
Joubert - Molar Tooth

Dandy Walker:

Radiologists love to nitpick and obsess over details. Usually, the more meaningless the detail the more ferocious the debate. Along those lines...

This is a “spectrum” that leads to much confusion and argument. For the purpose of the multiple choice tests I would just say that Dandy Walker = Absent Vermis.

The buzzword is “torcular-lambdoid inversion.” *The torcular is above the level of the lambdoid due to abnormally high tentorium.*



The following chart is just for the sake of addressing the ridiculousness of this posterior fossa situation. It ought to be very low yield for the CORE (which probably means it is high-yield). It may also be useful if you want to join in the debate.

Morphology (Most Severe -> Least Severe)					Features				
Ventriculocele” DWM Variant	Cystic Dilation of 4 th *	Complete or partial Agenesis of the Vermis	Enlarged Posterior Fossa PLUS erosion into the occipital bone-> encephalocele						
“Classic” DWM	Cystic Dilation of 4 th	Complete or partial Agenesis of the Vermis	Enlarged Posterior Fossa	Superiorly rotated vermian remnant					
“Variant” DWM	Partially obstructed 4 th Ventricle	Variable Hypoplasia of the Vermis (<i>less severe</i>)	Posterior Fossa NOT enlarged						
Persistent Blake Pouch	Cyst below and posterior to the vermis	Normal Vermis	Posterior Fossa NOT enlarged	The tentorium is elevated					
Mega Cisterna Magna	Retro-Cerebellar CSF Space > 10mm	Cerebellum Normal	Enlarged posterior fossa caused by enlarged cisterna magna						

Failure To Cleave

Holoprosencephaly (HPE): This is a midline cleaving problem with the brain failing to cleave into two separate hemispheres. The cleavage apparently occurs back to front (opposite of the formation of the corpus callosum), so in milder forms the posterior cortex is normal and the anterior cortex is fused.

It is classified as a spectrum:

Lobar Semilobar Alobar (most severe)

Lobar (<i>mild</i>)	Semi-Lobar	Alobar (<i>severe</i>)
Right and Left Hemispheres are separate (anterior/inferior frontal still sometimes fused)	Basic structure present but fused at the thalami. Posterior brain is normal.	There is a single large ventricle , with fusion of the thalami and basal ganglia .
May be limited to absent septum pellucidum	Olfactory tracts and bulbs are gone	No Falx. No corpus callosum.
Pituitary Problems are common		

The old saying “face predicts brain, but brain doesn’t predict face” is actually mostly true. In other words, monster Cyclops babies usually have some midline defect (holoprosencephaly). There are several other associations that are frequently shown and often testable:

HPE Associations:

- Single Midline Monster Eye
- Solitary Median Maxillary Incisor (MEGA-Incisor)
- Nasal Process Overgrowth leading to Pyriform Aperture Stenosis



: Meckel-Gruber Syndrome:

- Classic triad:
 - Holoprosencephaly,
 - Multiple Renal Cysts and
 - Polydactyly

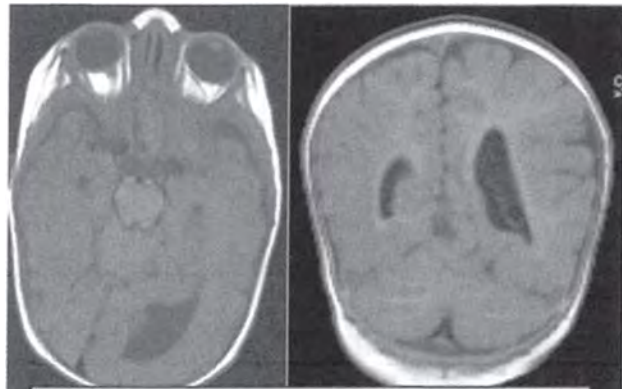
Anencephaly: This actually means “no -brain”. This is basically the extreme end of the Holoprosencephaly spectrum. You are going to have no brain (only a brainstem), no skull, and no scalp. *See the reproductive chapter for more on this.*

Failure to Migrate / Proliferate:

Hemimegalencephaly: Rare but unique (Aunt Minnie) malformation characterized by **enlargement of all or part/s of one cerebral hemisphere**. The cause of this condition is speculated to be problem of neuronal differentiation and cell migration in a single hemisphere. The affected hemisphere may have focal or diffuse neuronal migration defects, including areas of polymicrogyria, pachygyria, and heterotopia.

Here is the trick: Look at which side (the big side or the little side) has the ventriculomegaly.

- *Small Side + Big Ventricle = Atrophy (as might be seen with Rasmussen's Encephalitis)*
- *Big Side + Big Ventricle = Hemimegalencephaly*



! Hemimegalencephaly - Big Side Big Ventricle !

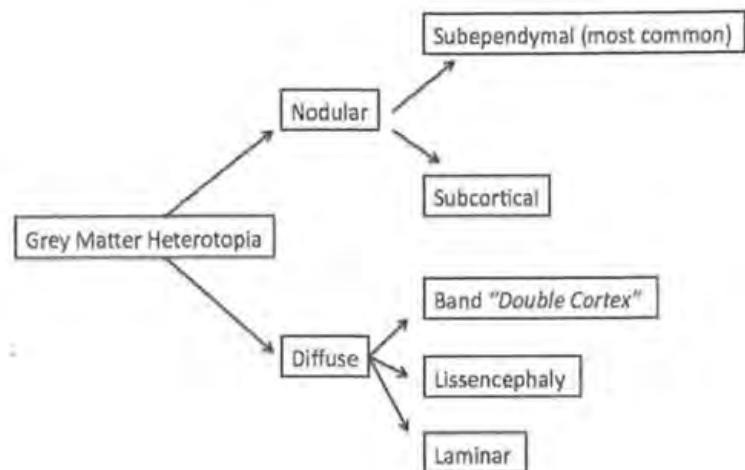
Lissencephaly-Pachygyria Spectrum: The spectrum of diseases that cause relative smoothness of the brain surface. Vocabulary includes agyria (no gyri), pachygyria (broad gyri) and lissencephaly (smooth brain surface).

You can think about it as either:

- Type I /Classic form = Smooth Brain. This results from arrest of migration. Buzzwords include “**figure 8**” , “**hours glass appearance**” , “**vertically oriented shallow Sylvian fissures**”. This one is associated with band heterotopias.
- Type II as a cobblestone brain. This results from over migration. There is not band heterotopia and the cortex is thinner than type I.

Grey Matter Heterotopias:

“Normal Neurons in Abnormal Locations” These guys can be grouped and then grouped and then subgrouped into groups.



They are associated with other congenital neurologic conditions. A random point of trivia: if you are trying to compare subependymal heterotopias with the subependymal tubers of TS, the tubers are usually higher on T2 signal than gray matter and are often calcified (except in early childhood).

Schizencephaly: Migrational disorder that results in a grey matter lined cleft that will extend through the entire hemisphere. It comes in two main flavors: (1) Closed Lip (20%) and (2) Open Lip (80%).

Highly Testable Associations:

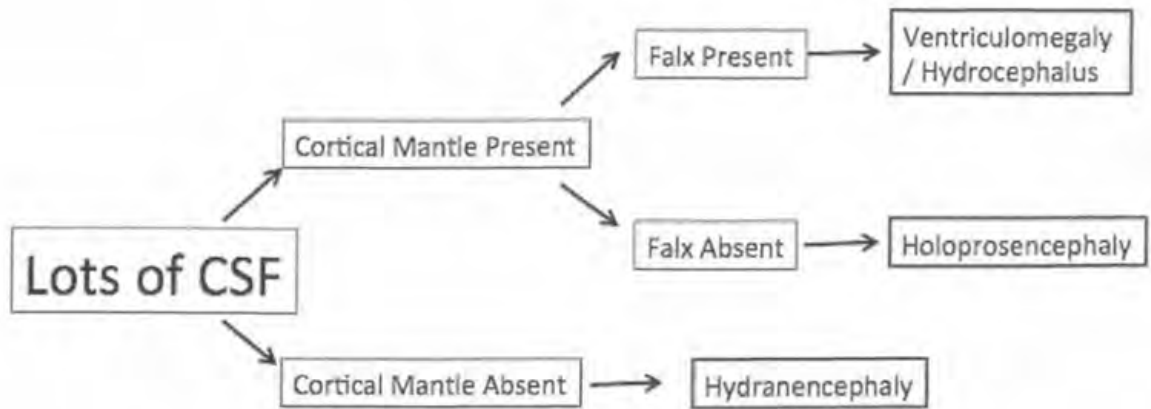
- *Optic Nerve Hypoplasia (30%)*
- *Absent Septum Pellucidum (70%)*
- *Epilepsy (demonic possession) 50-80%*

Classic THIS vs THAT: Porencephalic Cyst vs Open Lip Schizencephaly.

They look very similar, but the schizencephalic cleft should be lined with gray matter, and is a true malformation. The porencephalic cyst is a just a hole from a prior encephaloclastic event (ischemia). *Normal forming but massive insult makes you look like you didn't form.*

Hydranencephaly: Devastating condition characterized by destruction of the cerebral hemispheres. Basically this turns the skull into a bag of CSF. It is thought to be secondary to a vascular insult in utero (**think double MCA infarct**). This could be seen in the setting of a TORCH causing a necrotizing vasculitis (HSV does this). The key point is that you did have a normal brain, so you have a falx, but the cortical mantle is gone.

"Lots of csf" Brain Strategy



Herniation Syndrome Vocab

Cephaloceles - Herniation of the cranial contents through a defect in the skull.

- Meningoencephalocele - brain + Meninges
- Meningocele - just meninges (no brain)

Chiari Malformations:

Chiari malformation essentially occurs because of a mismatch between size and content of the posterior fossa.

Type 1 : These guys have headaches. Criteria is one cerebellar tonsil **more than 5mm below the foramen magnum**. This is often accompanied by crowding of the posterior fossa and you should look for syringohydromyelia (seen in about 50% of cases). As you age, the tonsils ascend, so 3mm below the foramen in a 90 year old would be abnormal. There are several associations but the one that is most often asked is with Klippel-Feil syndrome (congenital C-spine fusion).

Type 2 : This type is more complicated and there are like 20 findings. Hydrocephalus is found in more than 90% of cases. I'm going to try and pick the five I think they are most likely to ask or show.

- Myelomenigocele - Lumbar Spine
- Towering Cerebellum
- Tectal Plate Beaking
- Long Skinny 4th Ventricle (*elongated craniocaudally, short in other dimensions*)
— a normal 4th ventricle may suggest shunt malfunction
- Interdigitated Cerebral Gyri (most likely shown on axial CT, single image)



Tectal Beaking - Chiari 2

Type 3: Just think Chiari **II** + Encephalocele (either high cervical or low occipital)

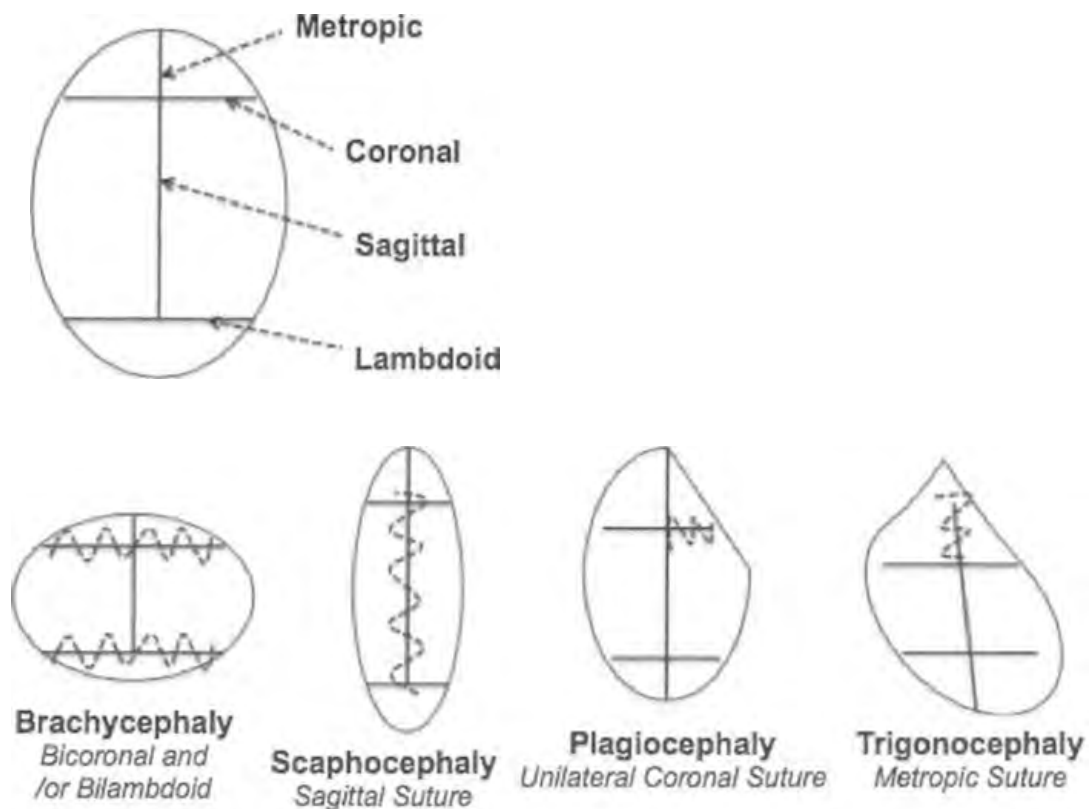
Craniosynostosis

Premature fusion of one or more of the cranial sutures can result in a weird shaped head. I have just a few high yield points regarding the subject. Scaphocephaly (Sagittal Suture) is the most common subtype and is often referred to as dolichocephaly. Brachycephaly (Coronal and/or Lambdoid) is often associated with syndromes.

Of course they are all zebras but a few are worth knowing:

- **Aperts:** *Brachycephaly + Fused Fingers*
- **Croutons:** *Usually Brachycephaly (but not always) + First Arch (Maxilla and Mandible Hypoplasia)*
- **Cleidocranial Dysostosis:** *Brachycephaly + Wormian Bones + Absent Clavicles*

Plagiocephaly can result from unilateral coronal or lambdoid suture fusion (frontal or occipital plagiocephaly). **Ipsilateral coronal fusion can elevate the superior orbital wall and cause a harlequin eye.**



High Yield Trivia / Buzzwords for Craniosynostosis:

- Most common type = sagittal (dolichocephaly or scaphocephaly)
- Sagittal Suture craniosynostosis almost always (80%) affects boys
- Coronal Suture Craniosynostosis affects more girls.
- “Harlequin Eye” = Unilateral Coronal Suture Craniosynostosis (lifts supra-orbital margin).
- Lambdoid Craniosynostosis favors the right side (70%)
- Turricephaly (the tower) is from both coronal and lambdoid fusion

Misc Peds Brain Conditions:

Enlarged extra-axial fluid spaces: Extra-axial fluid spaces are considered enlarged if greater than 5mm. It’s typically considered a benign finding related to immature arachnoid villi. It should resolve as the villi mature.

Choanal Atresia: This is a malformation of the choanal openings. Buzzwords are “*failure to pass NG tube*” and “*respiratory distress while feeding*” (neonates have to breathe through their noses). It’s usually unilateral (2/3). There are two different types: bony (90%), and membranous (10%). The appearance is a unilateral or bilateral posterior nasal narrowing, with thickening of the vomer. There are a bunch of syndromic associations: CHARGE, Crouzons, DiGeorge, Treacher Collins, and Fetal Alcohol Syndrome.

Piriform Aperture Stenosis - This can occur in isolation or with choanal atresia. The big thing to know is the **high association with hypothalamic-pituitary-adrenal axis dysfunction.**

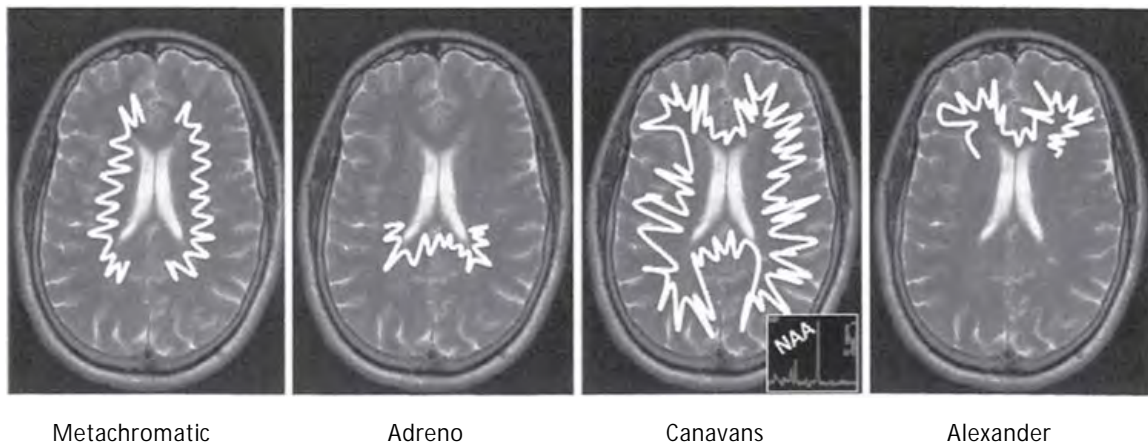
MELAS - This is a mitochondrial disorder with lactic acidosis and stroke like episodes. The SPECT question is always: increased lactate, decreased NAA.

Leukodystrophies: These are zebra disorders that affect the white matter in kids. If you see a brain MRI on a kid with jacked up white matter, you should be thinking about one of these guys. The distinction between them is totally academic, since they are all untreatable and fatal.

Here are my tricks:

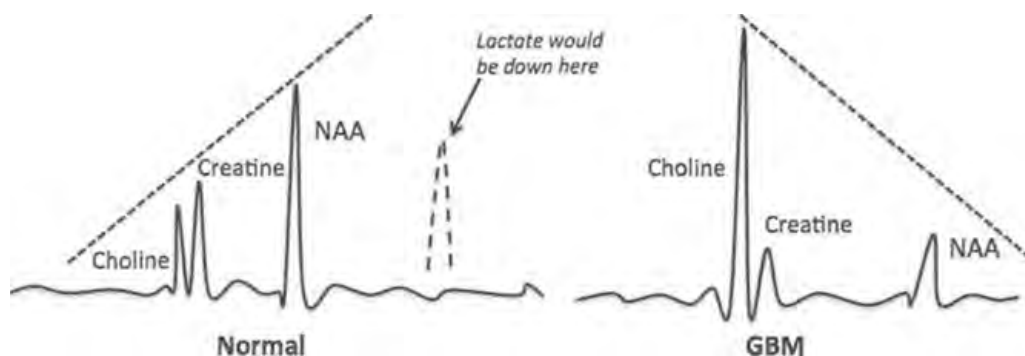
- *Canavans* is always shown with SPECT - with an elevated NAA.
- *Alexanders* has a big head (like Alexander the Great), and has frontal white matter involvement (frontal because of his personality disorder - metaphorically speaking).
- *Metachromatic* is the most common, and the buzzword is "tigroid." Tigroid refers to dark spots or stripes within the T2 bright demylinated periventricular white matter.

	Head Size	Age	Territory	Trivia
Metachromatic	Normal Head	Infantile form 1 -2 yo. Juvenile form 5-7	Diffuse white matter involvement, with tigroid appearance.	Most common Leukodystrophy. Deficiency of the enzyme arylsulfatase A.
Adreno Leukodystrophy	Normal Head	5-10 yo.	Symmetric occipital and splenium of corpus callosum white matter involvement.	Sex-linked recessive condition (peroxisomal enzyme deficiency) occurring only in boys.
Leigh disease	Normal Head	Less than 5 yo.	Focal areas of subcortical white matter. Basal ganglia and periaqueductal gray matter involvement.	Also called subacute necrotizing encephalomyelopathy. Mitochondrial enzyme defect.
Alexander disease	Big Head	Less than 1 yo.	Frontal white matter involvement.	
Canavan disease	Big Head	Less than 1 yo.	Diffuse Bilateral sub cortical U fibers.	Elevated NAA (MRS).



High Yield MR SPECT Trivia:

- The highest normal peak is NAA.
- The NAA peak will be super high with Canavans.
- Choline is elevated in anything that causes cell turnover (tumor, infarct, or inflammation).
- It's normal to see lactate elevated in the first hours of life.
- Myoinositol is elevated with Alzheimer's and low grade gliomas
- Alanine elevation is specific for Meningiomas
- Meningiomas do NOT have elevated NAA.
- Glutamine is elevated in Hepatic Encephalopathy
- High Grade Tumor = Choline Up, NAA down, Lactate and Lipids Up
- Low Grade Tumor = Choline Down, NAA down, Inositol Up
- Radiation Necrosis Pattern: Choline Down, NAA Down, Lactate Up



Notice that Choline, Creatine, and NAA fall in alphabetical order. That makes it easy to remember which is which

Section 2: Head and Neck

Temporal Bones:

Petrous Apex:

You can get 3 major types of pathology: (1) intrinsic stuff happening in the bone, (2) down-going processes from the intracranial compartment, and (3) up-going processes from the neck and sinuses.

Anatomic Variation: Variation can occur in the amount of pneumatization, marrow fat, bony continuity, and vascular anatomy.

- *Pneumatization:* - This is highly variable and can involve a large portion or small portion of the bone.
- *Asymmetric Marrow:* - Typically the petrous apex contains significant fat, closely following the scalp and orbital fat (T1 and T2 bright). When it's asymmetric you can have two problems (1) falsely thinking you've got an infiltrative process, when you don't and (2) overlooking a T1 bright thing (cholesterol granuloma) thinking it's fat. The key is to use STIR or some other fat saturating sequence.
- *Cephaloceles:* - A cephalocele describes a herniation of CNS content through a defect in the cranium. In the petrous apex they are a slightly different animal. They don't contain any brain tissue, and simply represent cystic expansion and herniation of the posterolateral portion of Meckel's cave into the superomedial aspect of the petrous apex. Describing it as a **herniation of Meckel's Cave would be more accurate**. These are usually unilateral and are classically described as "*smoothly marginated lobulated cystic expansion of the petrous apex*."
- *Variant Anatomy of the Carotid Artery.* - The 2 main variants to be aware of are the persistent stapedia artery, and aberrant internal carotid. For the purpose of the CORE exam the only things to know are **don't biopsy them** (*they aren't glomus tympanicum tumors*), and **they can give pulsatile tinnitus**.

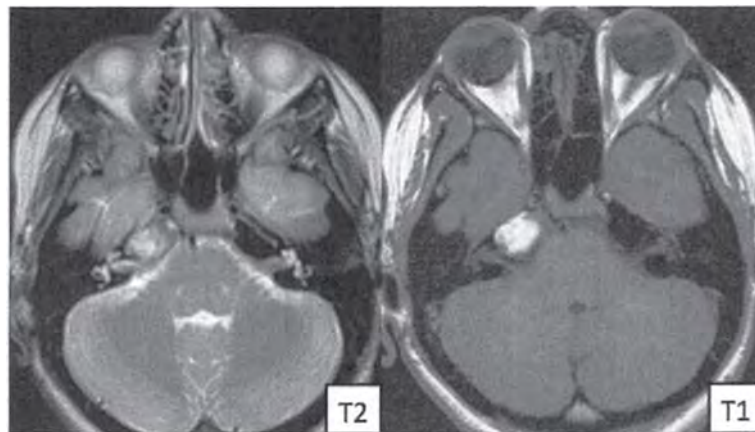
Carotid Artery Aneurysm - An aneurysm in the petrous portion is much less common than in the distal segments. They are usually asymptomatic and can be very large at diagnosis.

Inflammatory Lesions:

- ***Cholesterol Granuloma*** - The **most common primary petrous apex lesion**.

Mechanism is likely obstruction of the air cell, with repeated cycles of hemorrhage and inflammation leading to expansion and bone remodeling. The most common symptom is hearing loss. On CT the margins will be sharply defined. On MRI it's gonna be **T1 and T2 bright**, with T2 dark hemosiderin rim, and faint peripheral enhancement. The slow growing ones can be watched. The fast growing ones need surgery.

- **Key Point; Cholesterol Granuloma = T1 and T2 Bright.**



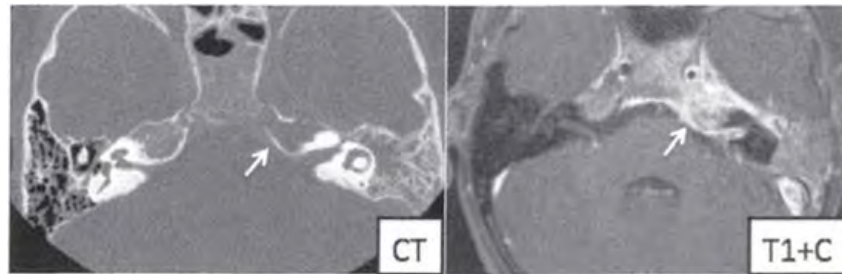
Cholesterol Granuloma = T1 and T2 Bright.

- ***Cholesteatoma*** - This is basically an epidermoid (ectopic epithelial tissue). They are congenital (not acquired) in the petrous apex. They are typically slow growing, and produce bony changes similar to cholesterol granuloma. The difference is their MRI findings; T1 dark, T2 bright, and restricted diffusion.

- **Key Point; Cholesteatoma = T1 Dark, T2 Bright, Restricted Diffusion**

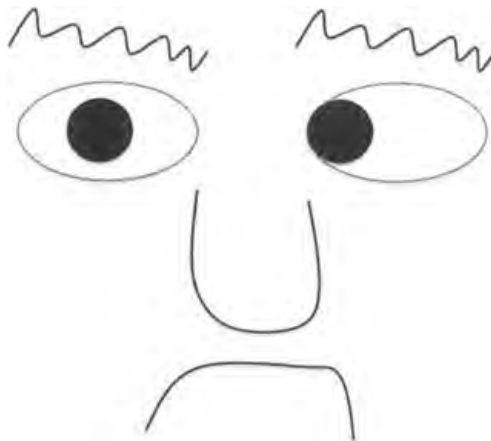
<i>Cholesterol Granuloma</i>	<i>Cholesteatoma</i>
T1 Bright	T1 Dark
T2 Bright	T2 Bright
Doesn't Restrict	Does Restrict
Smooth Expansile Bony Change	Smooth Expansile Bony Change

Apical Petrositis - Infection of the petrous apex is a rare complication of infectious otomastoiditis. It can have some bad complications if it progresses including osteomyelitis of the skull base, vasospasm of the ICA (if it involves the carotid canal), venous sinus thrombosis, and full on meningitis. In children it can present as a primary process. In adults it's usually in the setting of chronic otomastoiditis or recent mastoid surgery.



Apical Petrositis

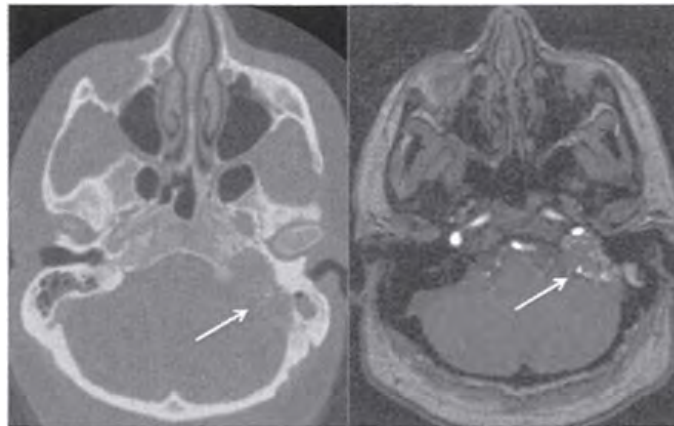
Gradenigo Syndrome - This is a complication of apical petrositis, when Dorello's canal (CN 6) is involved. They will show you (or tell you) that the patient has a **lateral rectus palsy**. The classic triad = otomastoiditis, face pain (trigeminal neuropathy), and lateral rectus palsy.



Clinical Correlation for Lateral Rectus Palsy

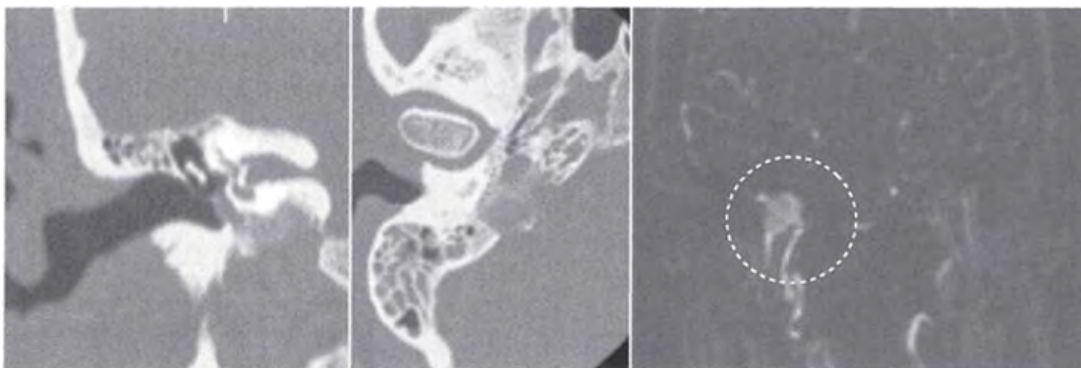
Tumors:

- *Endolymphatic Sac Tumor* - Rare tumor of the endolymphatic sac and duct. Although most are sporadic, when you see this tumor you should immediately think **Von-Hippel-Lindau**. They usually grow into the CPA. They almost always have **internal amorphous calcifications on CT**. There are T2 bright, with **intense enhancement**. They are **very vascular** often with **flow voids**, and **tumor blush** on angiography.



Endolymphatic Sac Tumor

- *Paraganglioma* - On occasion, paraganglioma of the jugular fossa (glomus jugulare or jugulotympanic tumors) can invade the occipital bone and adjacent petrous apex. As much as **40% of the time it's hereditary, and they are multiple**. The **most common presenting symptom is hoarseness** from vagal nerve compression. They are hypervascular, and heterogeneous **“salt and pepper”** appearing on **post contrast MRI, with flow voids**. They are FDG avid.

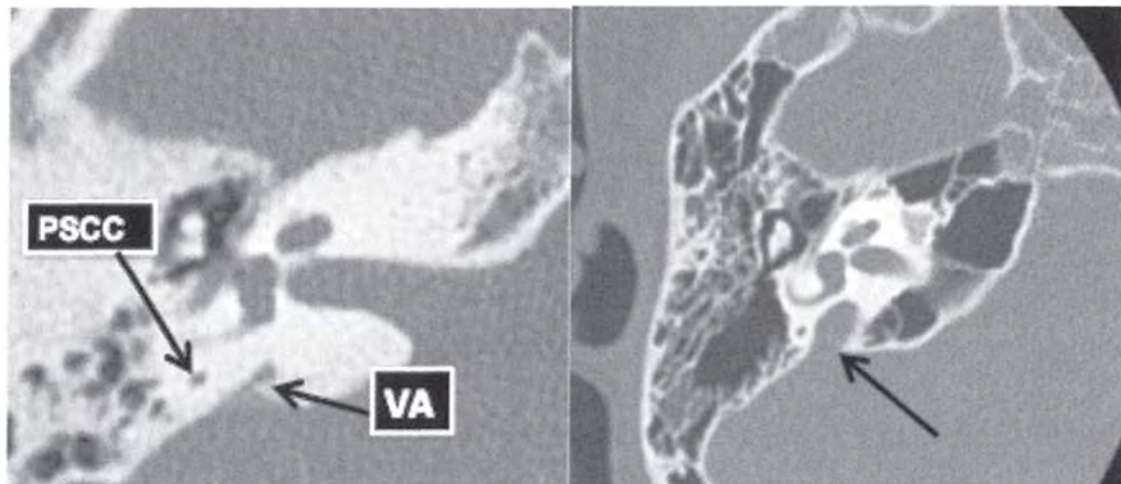


Paraganglioma - Glomus Jugulare

Inner Ear

Congenital

Large Vestibular Aqueduct Syndrome - The vestibular aqueduct is a bony canal that connects the vestibule (inner ear) with the endolymphatic sac. When this becomes enlarged ($> 1.5\text{mm}$) it is associated with **progressive sensorineural hearing loss**. As a point of trivia, the large vestibular aqueduct syndrome is associated with an **absence of the bony modiolus in more than 90% of patients**. This has an Aunt Minnie appearance. Supposedly this is the result of failure of the endolymphatic sac to resorb endolymph, leading to endolymphatic hydrops and dilation.



The normal VA is never larger than the adjacent PSCC

Enlarged Vestibular Aqueduct

Middle Ear

Otitis Media (OM) - This is a common childhood disease with effusion and infection of the middle ear. It's more common in children and patients with Down's Syndrome because of a more horizontal configuration of the Eustachian tube. It's defined as chronic if you have fluid persisting for more than six weeks. The complications are more of an issue (and more amenable to multiple choice questions).

Complications of OM:

- *Coalescent Mastoiditis* - erosion of the mastoid septae with or without intramastoid abscess
- *Facial Nerve Palsy* - Secondary to inflammation of the tympanic segment (more on this below).
- *Dural Sinus Thrombosis* - Adjacent inflammation may cause thrombophlebitis or thrombosis of the sinus. This in itself can lead to complications:
 - o *Venous Infarct*: This can occur secondary to dural sinus thrombosis
 - o *Otitic Hydrocephalus*: Venous thrombosis can affect resorption of CSF and lead to hydrocephalus.
- *Meningitis, and Labyrinthitis* - can both occur
- *Cholesteatoma* - This is a bunch of exfoliated skin debris growing in the wrong place. It creates a big inflammation ball which wrecks the temporal bone and the ossicles. The idea is basically this, you have two parts to the ear drum, a flimsy whippy part "Pars Flaccida", and a tougher part "Pars Tensa." The flimsy Flaccida is at the top, and the tensa is at the bottom. If you "acquire" a hole with some inflammation / infection involving the pars flaccida you can end up with this ball of epithelial crap growing and causing inflammation in the wrong place.

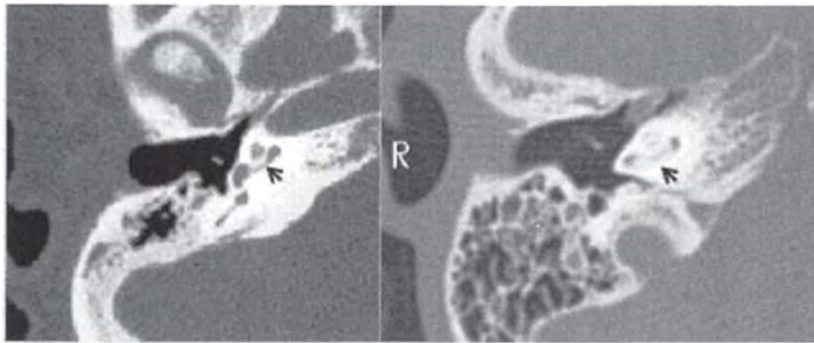
Cholesteatoma Order of Destruction predictable (and testable):

- (1) *The Scutum*
- (2) *The Ossicles (long process of the incus)*
- (3) *The Lateral Segment of the Semi-Circular Canal.*

Infections

Labyrinthitis - This is an inflammation of the membranous labyrinth, most commonly the result of a viral infection. The cochlea and semicircular canals will be shown enhancing on T1 post contrast imaging.

Labyrinthitis Ossificans - This is the sequella of prior infection (typically meningitis). You see it in kids (ages 2-18 months). They will show this on CT - with ossification of the membranous labyrinth. They also get sensorineural hearing loss.

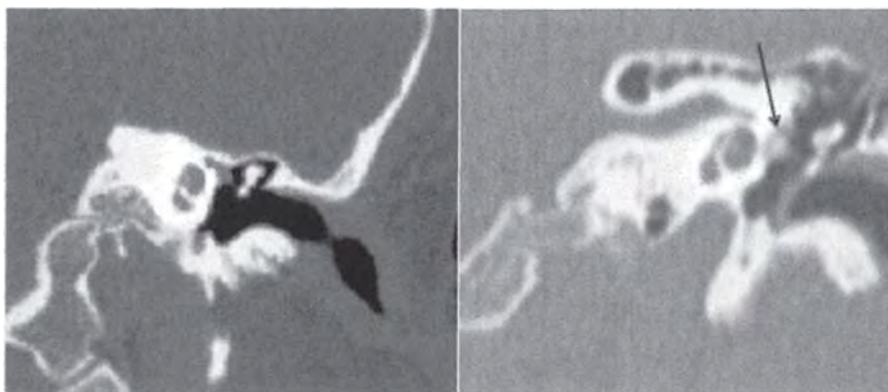


Normal

Labyrinthitis Ossificans

Otosclerosis (Fenestral and Retrofenestral): A better term would actually be “otospongiosis.” as the bone becomes more lytic (instead of sclerotic). When I say conductive hearing loss in an adult female, you say this.

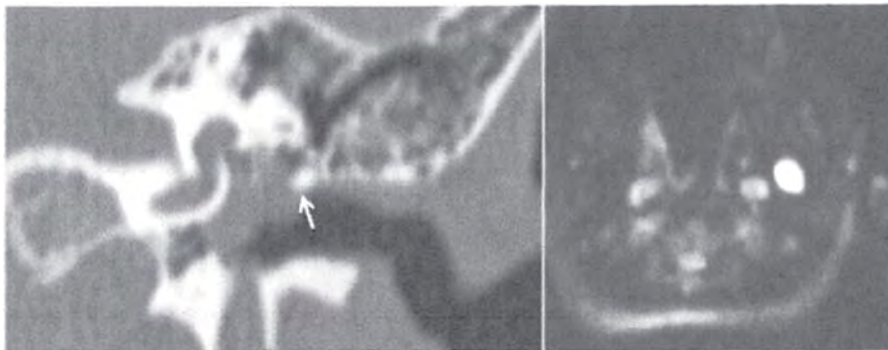
- *Fenestral* - This is **bony resorption anterior to the oval widow** at the fissula ante fenestram. If not addressed, the footplate will fuse to the oval window.
- *Retro-fenestral* - This is a **more severe** form, which has progressed to have **demineralization around the cochlea**. This form usually has a sensorineural component, and is **bilateral and symmetric nearly 100% of the time**.



Normal

Fenestral Otospongiosis

<i>Pars Flaccida Type</i>	<i>Pars Tensa Type:</i>
<ul style="list-style-type: none"> • <i>Acquired Types are more common - typically involving the pars flaccida. They grow into Prussak's Space</i> • <i>The Scutum is eroded early (maybe first) - considered a very specific sign of acquired cholesteatoma</i> • <i>The Malleus head is displaced medially</i> • <i>The long process of the incus is the most common segment of the ossicular chain to be eroded.</i> • <i>Fistula to the semi-circular canal most commonly involves the lateral segment</i> 	<ul style="list-style-type: none"> • <i>The inner ear structures are involved earlier and more often</i> • <i>This is less common than the Flaccida Type</i>

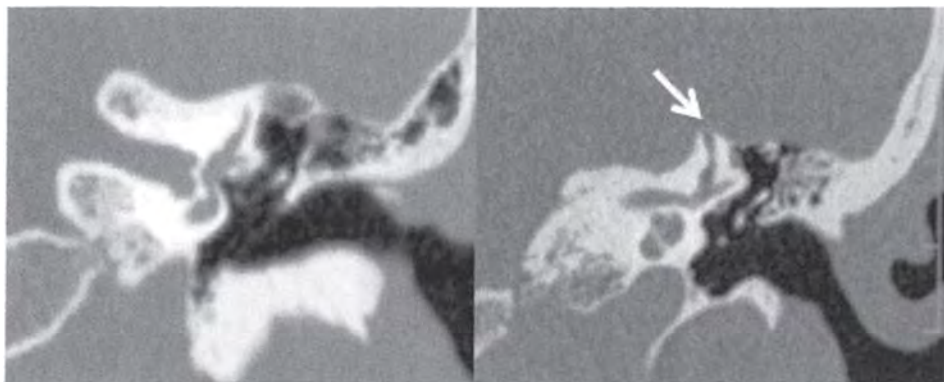


Pars Flaccida - Cholesteatoma

*Typical location with erosion of scutum (arrow)
They Restrict Diffusion*

Labyrinthine Fistula: This is a potential complication of cholesteatoma (can also be congenital or iatrogenic), in which there is a bony defect between the inner ear and tympanic cavity. **The lateral semicircular canal is most often involved.**

Superior Semicircular Canal Dehiscence: This is an Aunt Minnie. It's supposedly from long standing ICP although the most likely way this will be asked is either (1) what is it? with a picture or (2) **"Noise Induced Vertigo"** or "Tulio's Phenomenon." You are probably wondering who "Tulio" was. *Pietro Tullio was some mad scientist who drilled holes in the semicircular canals of pigeons then observed that they became off balance when he exposed them to sound. He also created a "pigeon rat" like Hugo Simpson did in the 1996 Simpsons Halloween Special (this is not confirmed).*



Normal Anatomy

Superior Semicircular
Canal Dehiscence

External Ear

Necrotizing External Otitis: This is a raging terrible infection of the external auditory canal. You are going to see swollen EAC soft tissues, probably with a bunch of small abscesses, and adjacent bony destruction. They always (95%) have diabetes and the causative agent is always (98%) *Pseudomonas*.

The Facial Nerve

There are only 2 or 3 things that are likely to be tested regarding the facial nerve.

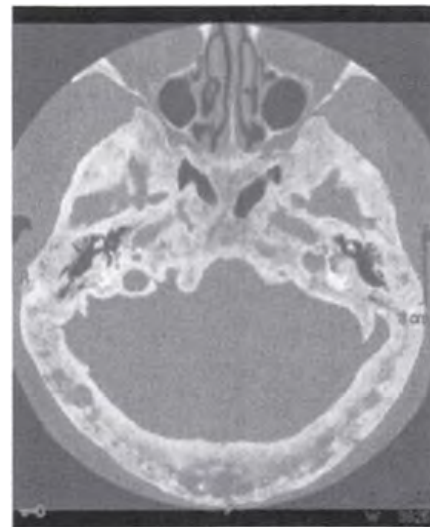
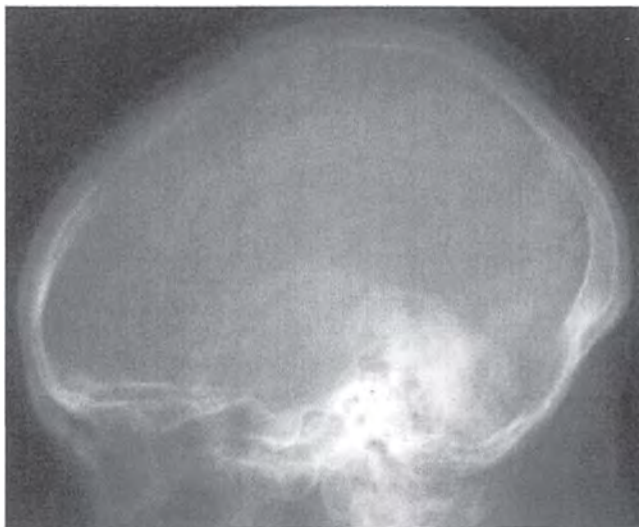
- (1) *Which parts normally enhance?* - It's easier to remember what does NOT. The cisternal, canalicular, and labyrinthine segments should NOT enhance. The remainder of the nerve - the intratemporal course - can enhance (tympanic, mastoid). Normal enhancement is due to the perineural venous plexus.
- (2) *What causes abnormal enhancement?* The big one is Bell's Palsy. Lyme's, Ramsay Hunt, and Cancer can do it too.
- (3) *When do you damage the facial nerve?* Usually the transverse T-Bone fracture (more than the longitudinal).

Skull Base

Pagets - This is discussed in great depth in the MSK chapter. Having said that, I want to remind you of the Paget skull changes. You can have osteolysis as a well-defined large radiolucent region favoring the frontal and occipital bones. The inner table is affected more than the outer table. The buzzword is **osteolysis circumscripta**.

Paget's Skull related complications:

- * Deafness is the most common complication
- Cranial Nerve Paresis
- * Basilar Invagination -> Hydrocephalus -> Brainstem Compression
- Secondary (high grade) osteosarcoma.



Pagets - *Osteolysis Circumscripta (lytic phase) Thickened Expanded Skull (Sclerotic Phase)*

Chordoma - This is a locally aggressive tumor that originates from the notochord (ergo, you can see it from the skull base to the sacrum). It is most commonly in the sacrum (second most common is the clivus).

Things to know:

- * Midline in the clivus
- * Projects posteriorly indenting the pons - "Thumb Sign"
- * T2 Bright

Chondrosarcoma - This is the main differential of the chordoma in the clivus. The thing to know is that it is **often lateral to midline** (*chordoma is midline*). These are also T2 bright.

Sinuses

Juvenile Nasal Angiofibroma (JNA) - Often you can get this one right just from the history. **Male teenager with nose bleeds** (obstruction is actually a more common symptom).

JNA Trivia to know:

- Mass is centered on the sphenoplatine foramen
- Expansion of the Pterygopalatine Fossa
- It's very vascular - and they may show an angiogram with a blush (during embolization therapy).
- Its primary vascular supply is from the ascending pharyngeal artery and/or internal maxillary artery

Antrochoanal Polyp - Also seen in young adults (30s-40s), this time with nasal congestion / obstruction symptoms. This one arises within the maxillary sinuses, and passes through and enlarges the sinus ostium (or accessory ostium). Buzzword is **“widening of the maxillary ostium.”** Classically, there is no associated bony destruction but instead smooth enlargement of the sinus. The polyp will extend into the nasopharynx. This thing is basically a monster inflammatory polyp with a thin stalk arising from the maxillary sinus.

Inverting Papilloma: This uncommon tumor has distinctive imaging features (which therefore make it testable). The **classic location is the lateral wall of the nasal cavity - most frequently related to the middle turbinate**. Impaired maxillary drainage is expected. **A focal hyperostosis tends to occur at the tumor origin. The MRI buzzword is “cerebriform pattern”** - which sorta looks like brain on T1 and T2. **Another high yield pearl is that 10% harbor a squamous cell CA.**

Esthesioneuroblastoma - This is a neuroblastoma of olfactory cells so it's gonna start at the cribriform plate. It classically has a dumbbell appearance with growth up into the skull and growth down into the sinuses, with a waist at the plate. There are often cysts in the mass. There is a bi-modal age distribution.

Things to know:

- *Dumbbell shape with wasting at the cribriform plate is classic*
- *Intracranial posterior cyst is a “diagnostic” look*
- *Octreotide scan will be positive - since it is of neural crest origin*

Squamous Cell/SNUC: Squamous cell is the **most common head and neck cancer**. The maxillary antrum is the most common location. It's highly cellular, and therefore low on T2. Relative to other sinus masses it enhances less. *SNUC* (the undifferentiated squamer), is the monster steroided up version of a regular squamous cell. They are massive and seen more in the ethmoids.

Path	Demographics	Typical Location	Trivia	Imaging Characteristics
Inverting Papilloma	40-70 M>F (4:1)	Lateral nasal wall centered at the middle meatus, with occasional extension into the antrum	40% show "entrapped bone" <i>Cerebriform Pattern</i> 10% Harbor a Squamous Cell CA	Cerebriform Pattern May have focal hyperostosis on CT
Esthesioneuroblastoma	Bimodal 20s & 60s	Dumbbell shaped with waist at the cribriform plate		AVID homogeneous enhancement
SNUC	Broad Range (30s-90s)	Ethmoid origin more common than maxillary	Lar. tvDically > 4cm on presentation	Fungating and Poorly defined Heterogeneous enhancement with necrosis
Squamous Cell CA	95% > 40 years old	Maxillary Antrum is involved in 80%	Most Common Malignancy of Sino-Nasal track	Aggressive Antral Soft Tissue Mass, with destruction of sinus walls Low signal on T2 (highly cellular) Enhances less than some other sinus malignancies
JNA (<i>Juvenile Nasopharyngeal Angiofibroma</i>)	Nearly Exclusively Male Rare < 8 or > 25	Origin in the Sphenopalatine Foramen (SPF)	Radiation alone cures in 80%	Enhancing mass arising from the SPF in adolescent male Dark Flow Voids on T1 Avidly Enhances
Sinonasal Lymphoma	Usually older, peak is 60s	Nasal Cavity > Sinuses	Highly variable appearance	Homogeneous mass in nasal cavity with bony destruction Low Signal on T2 (highly cellular)

Epistaxis - This is usually idiopathic, although it can be iatrogenic (picking it too much - or not enough). They could get sneaky and work this into a case of HHT. The most common location is the anterior septal area (Kiesselbach plexus), but because these are anterior they tend to be easy to compress manually. The posterior ones are less common (5%) but tend to be the ones that need angio. Most cases are given a trial of nasal packing. When that fails the N-IR team is activated. *The main supply to the posterior nose is the sphenopalatine artery (terminal internal maxillary artery) and tends to be the first line target.* Watch out for the variant anastomosis between the ECA and ophthalmic artery (you don't want to embolize the eye).

The Mouth

Floor of Mouth Dermoid / Epidermoid - There isn't a lot of trivia about these other than the buzzword and what they classically look like. The **buzzword** is “**sack of marbles**” - fluid sack with globules of fat. They are typically **midline**.

Ranula - This is a mucous retention cyst. They are typically **lateral**. There are two testable pieces of trivia to know: (1) **they arise from the sublingual gland / space**, and (2) use the word “**plunging**” **once it's under the mylohyoid muscle**.

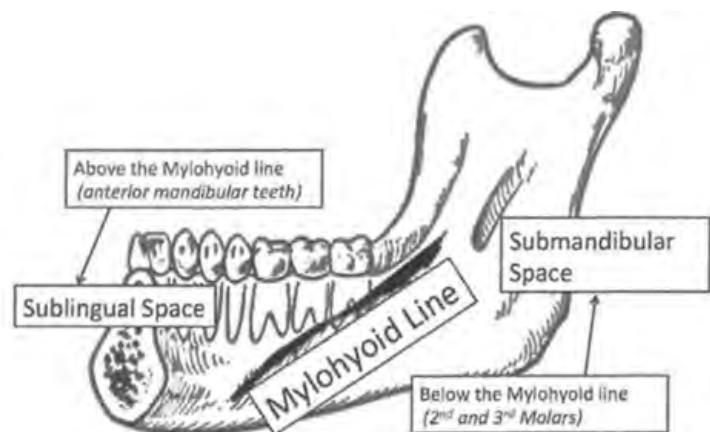
Torus Palatinus- This is a normal variant that looks scary. Because it looks scary some multiple choice writer may try and trick you into calling it cancer. **It's just a bony exostosis** that comes off the hard palate in the midline. Classic history “Grandma's dentures won't stay in.”

Sialolithiasis - Stones in the salivary ducts. The testable trivia includes: (1) **Most commonly in the submandibular gland duct (Wharton's)**, (2) can lead to an infected gland “sialoadenitis”, and (3) chronic obstruction can lead to gland fatty atrophy.

Odontogenic Infection - These can be dental or periodontal in origin. If I were writing a question about this topic **I** would ask three things. The first would be that infection is **more common from an extracted tooth** than an abscess involving an intact tooth.

The second would be that the **attachment of the mylohyoid muscle to the mylohyoid ridge dictates the spread of infection to the sublingual and submandibular spaces**. Above the mylohyoid line (anterior mandibular teeth) goes to the sublingual space, and below the mylohyoid line (second and third molars) goes to the submandibular space.

The third thing I would ask would be that an **odontogenic abscess is the most common masticator space “mass” in an adult**.



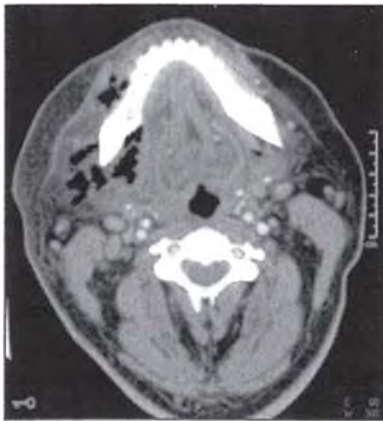
Ludwig's Angina - This is a super aggressive cellulitis in the floor of the mouth. If they show it, there will be gas everywhere. Trivia: most cases start with an odontogenic infection.

Osteonecrosis of the Mandible- The trivia is most likely gonna be etiology. Just remember it is related to prior radiation, licking a radium paint brush, or **bisphosphonate treatment**

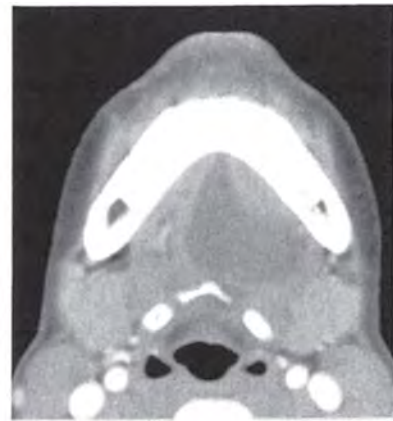
Cancer - Squamous cell is going to be the most common cancer of the mouth (and head and neck). In an older person think drinker and smoker. In a younger person think HPV. HPV related SCCs tend to be present with large necrotic level 2a nodes (don't call it a brachial cleft cyst!). *Classic scenario = young adult with new level II neck mass = HPV related SCC.*

Thyroglossal Duct Cyst - This can occur anywhere between the foramen cecum (the base of the tongue) and the thyroid gland. They are usually found in the midline at or above the hyoid. It looks like a thin walled cyst. Further discussion in the endocrine chapter.

Classic Mouth Pictures:



Ludwig's Angina Osteonecrosis
of Jaw



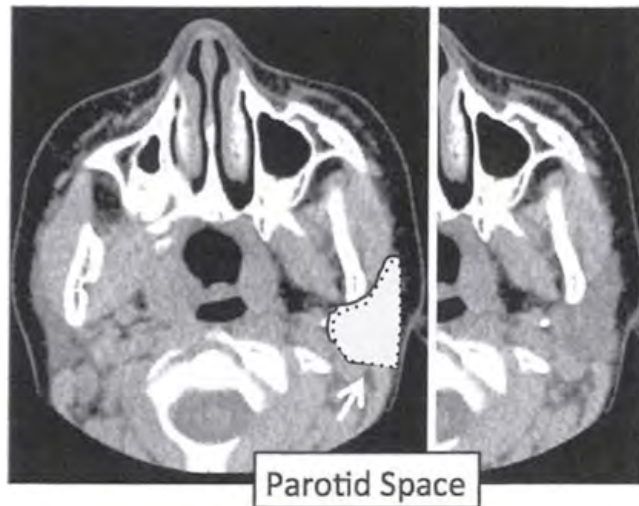
Ranula

Suprahyoid Soft Tissue Neck

The suprahyoid neck is usually taught by using a “spaces” method. This is actually the best way to learn it. What space is it? What is in that space? What pathology can occur as the result of what normal structures are there. Example: lymph nodes are there - thus you can get lymphoma or a met.

Parotid Space:

The parotid space is basically the parotid gland, and portions of the facial nerve. You can't see the facial nerve, but you can see the retromandibular vein (which runs just medial to the facial nerve). Another thing to know is that the parotid is the only salivary gland to have lymph nodes, so pathology involving the gland itself, and anything lymphatic related is fair game.



Pathology:

Pleomorphic Adenoma (benign mixed tumor) - This is the most common major (*and minor*) salivary gland tumor. It occurs most commonly in the parotid, but can occur in the submandibular, or sublingual glands. 90% of these tumors occur in the superficial lobe. They are commonly T2 bright, with a rim of low signal. **They have a small malignant potential** and are treated surgically.

- *Superficial vs Deep:* Involvement of the superficial (lateral to the facial nerve) or deep (medial to the facial nerve) lobe is critical to the surgical approach. A line is drawn connecting the lateral surface of the posterior belly of the digastric muscle and the lateral surface of the mandibular ascending ramus to separate superficial from deep.
- Apparently, if you resect these like a clown you can spill them, and they will have a massive ugly recurrence.

Warthins: This is the second most common benign tumor. This one **ONLY** occurs in the parotid gland. This one is **usually cystic, in a male, bilateral (15%), and in a smoker**. As a point of total trivia, this tumor **takes up pertechnetate** (it's basically the only tumor in the parotid to do it, *ignoring the ultra rare parotid oncocytoma*).

Mucoepidermoid Carcinoma - This is the **most common malignant tumor of minor salivary glands**. The general rule is - the smaller the gland the more common the malignant tumors, the bigger the gland the more common the benign tumors. There is a variable appearance based on the histologic grade. There is an association with radiation.

Adenoid Cystic Carcinoma - This is another malignant salivary gland tumor, which favors minor glands but can be seen in the parotid. The number one thing to know is perineural spread. This tumor likes perineural spread. **When 1 say adenoid cystic you say perineural spread.**

Lymphoma - Because the parotid has lymph nodes (it's the only salivary gland that does), you can get lymphoma in the parotid (primary or secondary). If you see it and it's bilateral, you should think Sjogrens. Sjogrens patients have a big risk (like 1000x) of parotid lymphoma. Like lymphoma is elsewhere in the body, the appearance is variable. You might see bilateral homogeneous masses. For the purposes of the CORE exam, **just knowing you can get it in the parotid (primary or secondary) and the relationship with Sjogrens is probably all you need.**

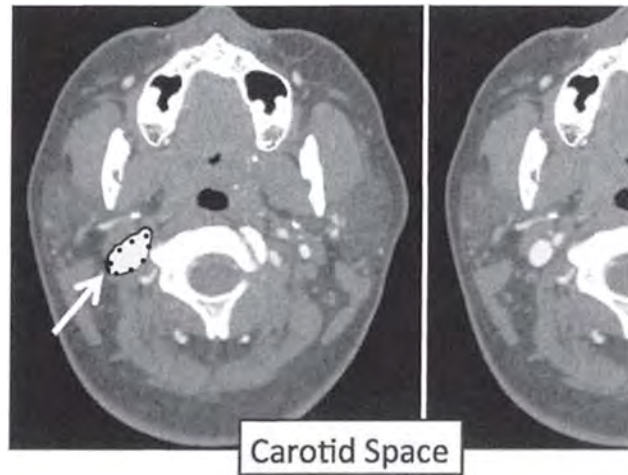
Other Parotid Trivia:

- *Acute Parotitis*: Obstruction of flow of secretions is the most common cause. They will likely show you a stone (or stones) in Stensen's duct, which will be dilated. The stones are calcium phosphate. Post infectious parotitis is usually bacterial. Mumps would be the most common viral cause. As a point of trivia, sialography is contraindicated in the acute setting.
- *Benign Lymphoepithelial Disease*: You have **bilateral mixed solid and cystic lesions** with diffusely enlarged parotid glands. This is **seen in HIV**. The condition is painless (unlike parotitis - which can enlarge the glands).
- *Sjogrens* - Autoimmune lymphocyte induced destruction of the gland. "Dry eyes and Dry Mouth." Typically seen in women in their 60s. **Increased risk** (like 1000x) risk of non-Hodgkins MALT type **lymphoma**. There is a **honeycombed appearance of the gland**.

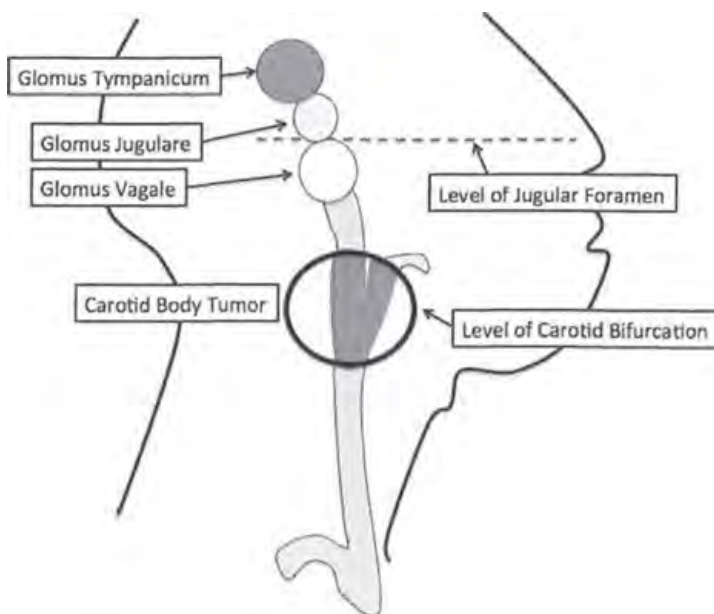
Carotid Space:

This space includes the carotid artery, the jugular vein, portions of CN 9, CN 10, CN 11, and the internal jugular chain lymph nodes.

Lymph Nodes = Metastatic lesions from squamous cell cancer frequently involve this area.



Paragangliomas: There are three different ones worth knowing about - based on location. The imaging features are the same. They are **hypervascular (intense tumor blush)**, with a **“Salt and Pepper” appearance on MRI** from all the heterogeneous stuff and flow voids. They can be multiple and bilateral in familial conditions. **¹¹¹In- octreotide accumulates in these tumors** (receptors for somatostatin).



Carotid Body Tumor = Carotid Bifurcation {*Splaying ICA and ECA*)

Glomus Jugulare = Skull Base {*often with destruction of jugular foramen*)

Glomus Vagale = Above Carotid Bifurcation, but below the Jugular Foramen

Glomus Tympanicum = Confined to the middle ear. Buzzword is “*overlying the cochlear promontory.*”

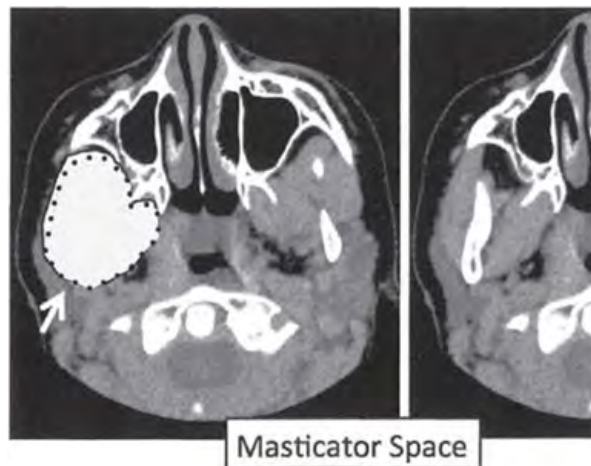
Schwannoma - Can involve the 9, 10, 11, and even 12th CN.

Schwannoma	Paraganglioma
Not all that vascular on Angio	Hypervascular (tumor blush on angio)
No Salt and Pepper	Salt and Pepper Look on MRI
NOT ¹¹¹ In- octreotide avid	¹¹¹ In- octreotide avid
No Flow Voids (target sign)	Flow Voids

Lemierre syndrome - This is a thrombophlebitis of the jugular veins with distant metastatic sepsis (usually septic emboli in the lung). It's found in the setting of oropharyngeal infection (pharyngitis, tonsillitis, peri tonsillar abscess), or recent ENT surgery.

Masticator Space:

As the name implies this space contains the muscles of mastication (masticator, temporalis, medial and lateral pterygoids). Additionally, you have the angle and ramus of the mandible, plus the inferior alveolar nerve. A trick to be aware of is that the space extends superiorly along the side of the skull via the temporalis muscle. So, aggressive neoplasm or infection may ride right up there.



Odontogenic Infection - In an adult this is the most common cause of a masticator mass. If you see a mass here, the next move should be to look at the mandible on bone windows. Just in general, you should be on the look out for spread via the pterygopalatine fossa to orbital apex and cavernous sinus. The relationship with the mylohyoid makes for good trivia - as discussed above.

Sarcomas - In kids, you can run into nasty angry masses like Rhabdomyosarcomas. You can also get sarcomas from the bone of the mandible (chondrosarcoma favors the TMJ).

Cavernous Hemangiomas - These can also occur, and are given away by the presence of phleboliths. Venous or lymphatic malformations may involve multiple compartments / spaces.

**Congenital stuff and Aggressive Infection/Cancer tends to be trans-spatial.*

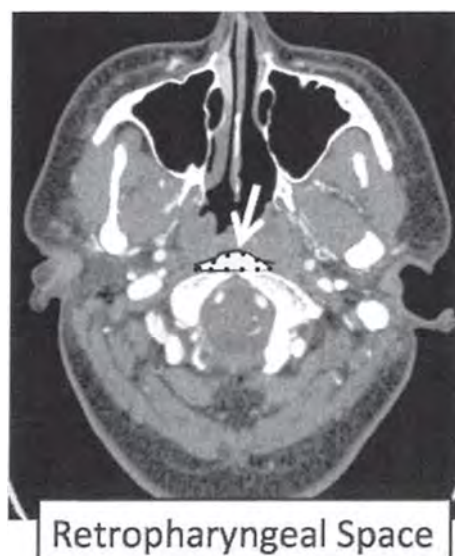
Perineural Spread - You can have perineural spread from a head and neck primary along the 5th CN. You should look at foramen ovale and make sure it is not expanded. When I say “perineural spread” you should think two things: **(1) adenoid cystic** minor salivary tumor and **(2) melanoma**.

Nerve Sheath Tumors - Since you have a nerve, you can have a schwannoma or neurofibroma of V3.

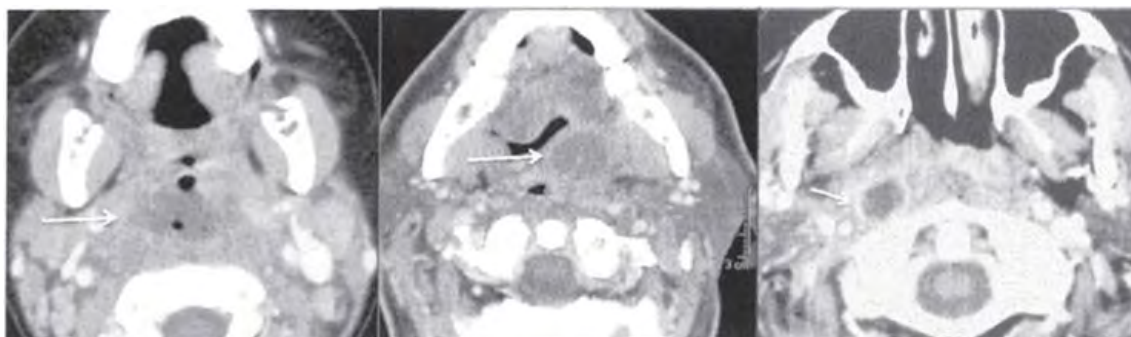
Retropharyngeal Space /Danger Space

Behind the alar fascia, this is a potential midline space which can be used as a major highway extending superior to the skull base and/or inferior down to approximately T3. This is the so called “danger space”, and can dump infection / cancer right into the mediastinum (that’s bad).

Infection - Involvement of the retropharyngeal space most often occurs from spread from the tonsillar tissue. You are going to have enhancing soft tissue and stranding in the space. You should evaluate for spread of infection into the mediastinum.



Necrotic Nodes - **Squamous cell mets** or suppurative infection to the lateral retropharyngeal nodes of Rouviere. Papillary thyroid cancer can also met here. Lymphoma can involve these nodes, but won’t be necrotic until treated.



Retropharyngeal Abscess Peritonsillar Abscess Suppurative Node of Rouviere

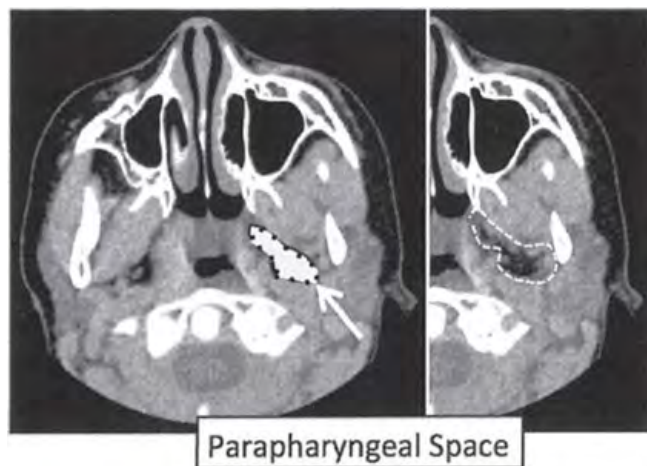
Neck Infection Syndromes

Grisel's Syndrome - Torticollis with atlanto-axial joint inflammation seen in H&N surgery or retropharyngeal abscess

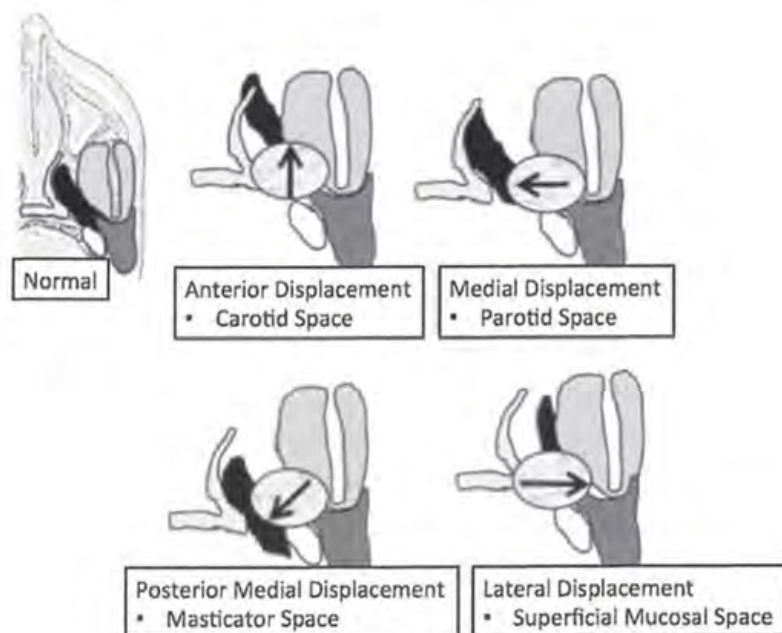
Lemierre's Syndrome - URI / Neck injection or recent ENT surgery leads to jugular vein thrombosis and septic emboli. Buzzword bacteria = "Fusobacterium Necrophorum"

Parapharyngeal Space

The parapharyngeal space is primarily a ball of fat with a few branches of the trigeminal nerves, and the pterygoid veins. The primary utility of the space is when it is displaced (discussed below). Mets and infections can directly spread into this space (squamous cell cancer from tonsils, tongue, and larynx). Cancer and infection can spread rapidly in a vertical direction through this fat.



The parapharyngeal space is bordered on four sides by different spaces. If you have a mass dead in the middle, it can be challenging to tell where it's coming from. Using the displacement of fat, you can help problem solve. Much more important than that, this lends itself very well to multiple choice.

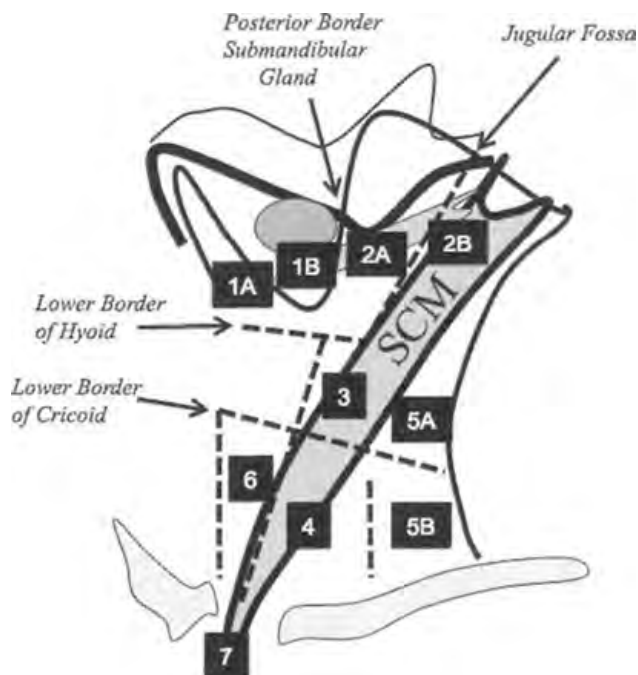


Parotid Mass Pushes
Medially (PMPM)

Squamous Cell Cancer

When you are talking about head and neck cancer, you are talking about squamous cell cancer. Now, this is a big complex topic and requires a fellowship to truly understand / get good at. Obviously, the purpose of this book is to prepare you for multiple choice test questions not teach you practical radiology. If you want to actually learn about head and neck cancer for a practical sense you can try and find a copy of Harnsberger's legendary handbook (which has been out of print for 20 years). A more modern alternative is probably Raghavan's Manual of Head and Neck Imaging. Now, for the trivia....

Lymph node anatomy:



Testable Trivia:

Anterior Belly of Digastric separates 1A from 1B

Stylohyoid muscle
(posterior submandibular gland) separates 1B from 2A

Spinal Accessory Nerve
(jugular) separates 2A from 2B

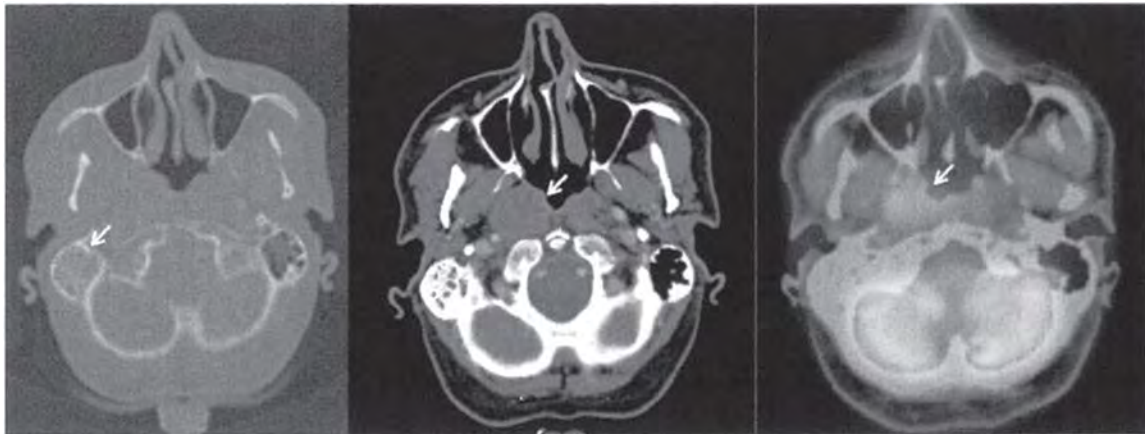
Vertical borders:

2- 3 = Lower Hyoid

3- 4 = Lower Cricoid

Floor of the Mouth SCC: I touched on this once already. Just remember smoker/drinker in an old person. HPV in a young person. Necrotic level 2 nodes can be a presentation (not a brachial cleft cyst).

Nasopharyngeal SCC: This is more common in Asians and has a bi-modal distribution: group 1 (15-30) typically Chinese, and group 2 (> 40). Involvement of the parapharyngeal space results in worse prognosis (compared to nasal cavity or oropharynx invasion). The **most common location is the Fossa of Rosenmuller (FOR)**. If you see (1) a unilateral mastoid effusion, or (2) a pathologic retropharyngeal node look in the FOR. The “*earliest sign*” of nasopharyngeal SCC is the effacement of the fat within the FOR. If you see a supraclavicular node then you should look closely at the bones for mets (especially the clivus).



Unilateral Mastoid Effusion Fossa of Rosenmuller Mass

PET Avid

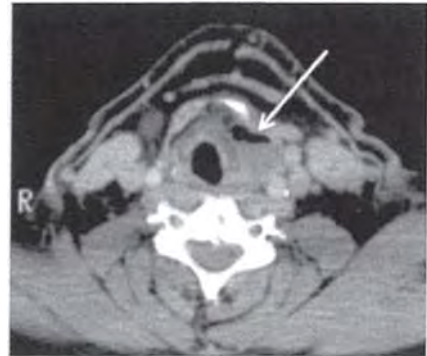
Laryngeal SCC: The role of the Radiologist is not to make the primary cancer diagnosis here, but to assist in staging. Laryngeal cancers are subdivided into (a) supraglottic, (b) glottic, and (c) infraglottic types. “Transglottic” would refer to an aggressive cancer that crosses the laryngeal ventricle.

Things to know about laryngeal SCC:

- Glottic SCC has the best outcome (least lymphatics), and is the most common (60%)
- Subglottic is the least common (5%), and can be clinically silent till you get nodes
- Subglottic tumors are often small compared to nodal burden
- Fixation of the cords indicates at least a T3 tumor
- The only reliable sign of cricoid invasion is tumor on both sides of the cartilage (irregular sclerotic cartilage can be normal).
- Invasion of the cricoid cartilage is a contraindication to all types of laryngeal conservation surgery (cricoid cartilage is necessary for postoperative stability of the vocal cords).
- Paraglottic space involvement makes the tumor T3 and “transglottic.” This is best seen in coronals.

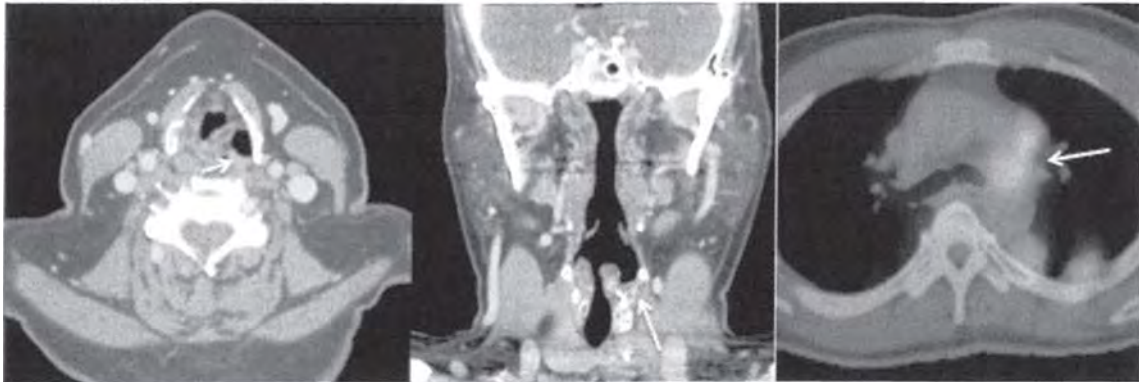
Infrahyoid Soft Tissue Neck

Laryngocele: When the laryngeal saccule dilates with fluid or air you call it a laryngocele. The testable trivia is that they can be associated with a tumor (causing obstruction).



Laryngocele

Vocal Cord Paralysis: The **affected side will have an expanded ventricle** (it's the opposite side with a cancer). If you see it on the left, a good “next step” question would be to look at the chest (for recurrent laryngeal nerve involvement at the AP window).

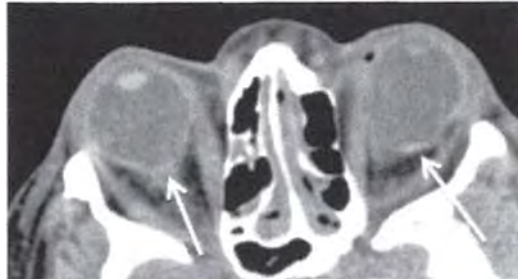


Vocal Cord Paralysis - Ipsilateral Expanded Ventricle AP Window PET Avid Node

Orbit

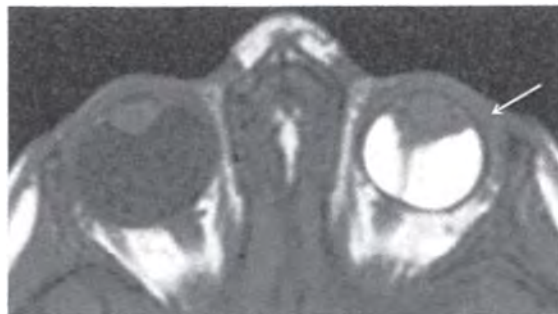
Congenital

Coloboma: This is a focal discontinuity of the globe (failure of the choroid fissure to close). They are usually posterior. If you see a unilateral one - think sporadic. **If you see bilateral ones - think CHARGE** (coloboma, heart, GU, ears).



Bilateral Coloboma - CHARGE Syndrome

Persistent Hyperplastic Primary Vitreous (PHPV) - This is a failure of the embryonic ocular blood supply to regress. It can lead to retinal detachment. The classic look is a **small eye (microphthalmia) with increased density of the vitreous**. No calcification.



Retinal Detachment in the setting of PHPV

Coat's Disease - The cause of this is a retinal telangiectasis, that results in leaky blood and subretinal exudate. It can lead to retinal detachment. It's seen in young boys and typically unilateral. The key detail is that it is **NOT CALCIFIED** (retinoblastoma is).

Retinal Detachment - This can occur secondary to PHPV or Coats. It can also be caused by trauma, sickle cell, or just old age. The imaging finding is a "V" or "Y" shaped appearance due to lifted up retinal leaves and subretinal fluid (as seen in the PHPV case above).

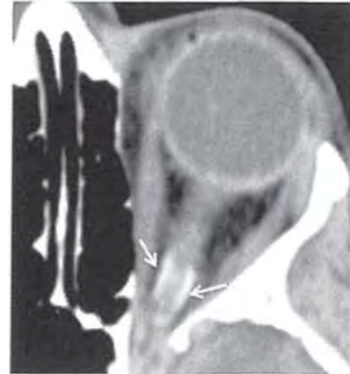
Globe Size - A Source of Possible Trivia

- | | |
|---------------------------------------|--|
| • <i>Retinoblastoma</i> - Normal Size | • <i>PHPV</i> - Small Size (<i>Normal Birth Age</i>) |
| • <i>Coats</i> - Normal Size | • <i>Retinopathy of Prematurity</i> - Bilateral |
| • <i>Toxocariasis</i> - Normal Size | Small |

Orbital Tumors:

Optic Nerve Glioma: These almost always (90%) occur under the age of 20. You see expansion / enlargement of the entire nerve. If they are bilateral you think about **NF-1**. They are most often WHO grade 1 *Pilocystic Astrocytomas*. If they are sporadic they can be GBMs and destroy you.

Optic Nerve Sheath Meningioma: The buzzword is “tram-track” calcifications. Another buzzword is “doughnut” appearance, with **circumferential enhancement around the optic nerve**.



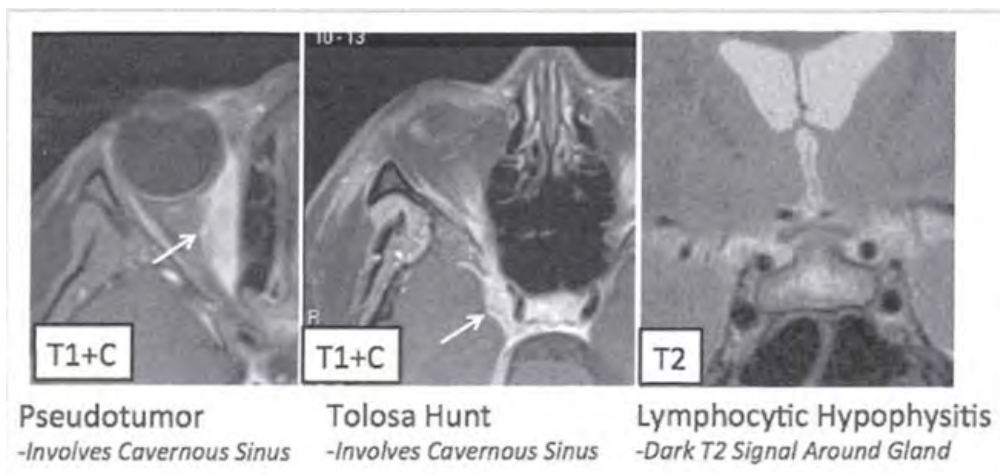
"Tram-Track" = Meningioma

IgG4 - Orbit

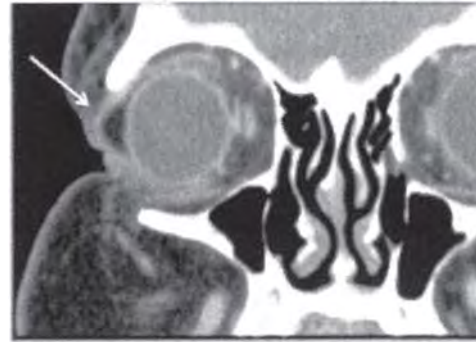
Orbital Pseudotumor: This is one of those IgG4 idiopathic inflammatory conditions that involves the extraocular muscles. It looks like an expanded muscle. The things to remember are that this thing is **painful**, unilateral, it **most commonly involves the lateral rectus** and it **does NOT spare the myotendinous insertions**. Remember that Graves does not cause pain, and does spare the myotendinous insertions. It gets better with steroids. It's classically T2 dark.

Tolosa Hunt Syndrome: This is histologically the same thing as orbital pseudotumor but instead involves the cavernous sinus. It is painful (just like pseudotumor), and presents with multiple cranial nerve palsies. It responds to steroids (just like pseudotumor).

Lymphocytic Hypophysitis: This is the same deal as orbital pseudotumor and Tolosa Hunt, except it's the pituitary gland. Just think enlarged pituitary stalk, in a post partum / 3rd trimester woman. It looks like a pituitary adenoma, but it classically has a T2 dark rim.



Dermoid: This is the most common benign congenital orbital mass. It's usually **superior and lateral**, arising from the frontozygomatic suture, and presenting in the first 10 years of life. It's gonna have fat in it (like any good dermoid). The location is classic.



Orbital Dermoid - *Classic Location*

Rhabdomyosarcoma - *Most common extraocular orbital malignancy in children* (dermoid is most common benign orbital mass in child). It's still rare as hell.

Metastatic Neuroblastoma - This has a very classic appearance of **"Raccoon Eyes"** on physical exam. The *classic location is periorbital tumor infiltration with associated proptosis*. Don't forget a basilar skull fracture can also cause Raccoon Eyes... so clinical correlation is advised.

Metastatic Breast Cancer - This is classic gamesmanship here. The important point to know is that unlike primary orbital tumors that are going to cause proptosis, classically the **breast cancer met causes a desmoplastic reaction and enophthalmos**.

Lymphoma - There is an association with **Chlamydia Psittaci** (the bird fever thing) and MALT lymphoma of the orbit. It usually involves the upper outer orbit - closely associated with the lacrimal gland. It will enhance homogeneously and restricts diffusion - just like in the brain.

Globe Tumors:

Melanoma - This is the **most common intra-ocular lesion in an adult**. If you see an enhancing soft tissue mass in the back of an adult's eye this is the answer. I can only think of three ways you could ask about this: (1) show a picture - what is it?, (2) ask what the most common intra-ocular lesion in an adult is, or (3) ask the buzzword *"collar button shaped"* - which is related to Bruch's membrane.

Retinoblastoma - This is the most common primary malignancy of the globe. The step 1 question is RB suppressor gene (chromosome 13). That's the same chromosome osteosarcoma patients have issues with and why these guys are at increased risk of facial osteosarcoma after radiation. **If you see calcification in the globe of a child - this is the answer.** It's T2 bright (Coats and PHPV are T2 dark). It's **usually seen before age 3** (rare after age 6). The trivia is gonna be where else it occurs. They can be bilateral (both eyes - 30%), *trilateral* (both eyes, and the pineal gland), and *quadrilateral* (both eyes, pineal, and suprasellar).

Orbital Vascular Malformations:

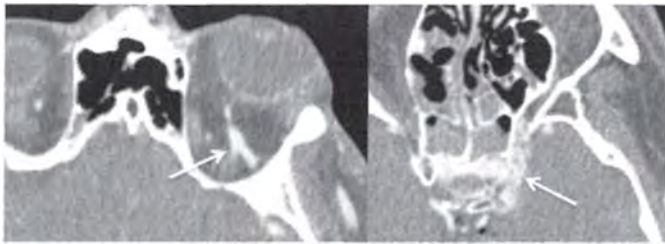
Lymphangioma - These are actually a mix of venous and lymphatic malformations. They are ill-defined and lack a capsule. The usual distribution is infiltrative (**multi-spatial**) involving, pre septal, post septal, extraconal, and intraconal locations. **Fluid-Fluid levels** are the money shot, and the most likely finding to be shown by someone writing a multiple choice test question. They do NOT increase with valsalva.



Lymphangioma
- *Fluid Fluid Levels*

Varix - These occur secondary to weakness in the post capillary venous wall (gives you massive dilation of the valveless orbital veins). Most likely question is going to pertain to the fact that **they distend with provocative maneuvers** (Valsalva, hanging head, etc...). Another piece of trivia is that they are the *most common cause of spontaneous orbital hemorrhage*. They can thrombose and present with pain.

Carotid-Cavernous Fistula: These come in two flavors: (1) Direct - which is secondary to trauma, and (2) Indirect - which just occurs randomly in post menopausal women. The direct kind is a communication between the intracavernous ICA and cavernous sinus. The indirect kind is usually a dural shunt between meningeal branches of the ECA and Cavernous Sinus.



Carotid-cavernous Fistula - *Prominent left superior ophthalmic vein, prominent left cavernous sinus with proptosis.*

Pulsatile Exophthalmos *The Buzzword*

C-C Fistula is probably the most common cause.

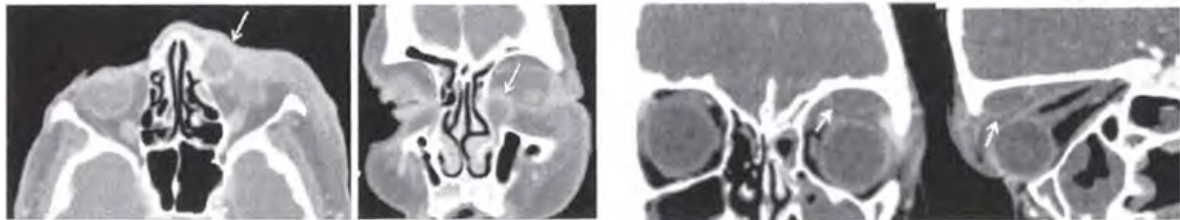
NF-1 can also cause it, from *sphenoid wing dysplasia*.

Orbital Infection:

Pre-Septal/ Post Septal Cellulitis - The location of orbital infections are described by their relationship to the orbital septum. The testable trivia is probably (1) that the orbital septum originates from the periosteum of the orbit and inserts in the palpebral tissue along the tarsal plate, (2) that pre-septal infections usually start in adjacent structures likely teeth and the face, and that (3) post-septal infections are usually from paranasal sinusitis.

Dacryocystitis - This is inflammation and dilation of the lacrimal sac. It has an Aunt Minnie look, with a well circumscribed round rim enhancing lesion centered in the lacrimal fossa. The etiology is typically obstruction then bacterial infection (staph and strep).

Orbital Subperiosteal Abscess: If you get inflammation under the periosteum it can progress to abscess formation. This is usually associated with ethmoid sinusitis. This also has a very classic look.



Dacryocystitis

Orbital Subperiosteal Abscess

Misc:

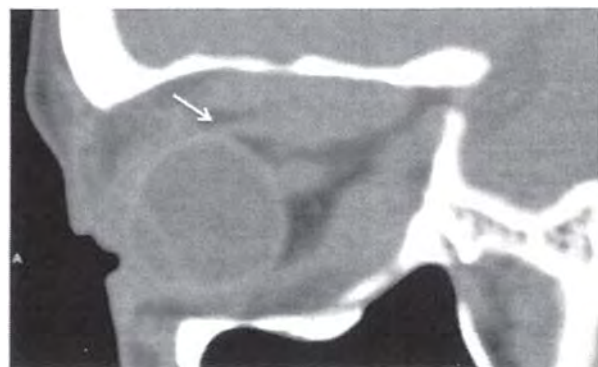
Optic Neuritis: There will be **enhancement of the optic nerve**, *without enlargement* of the nerve/sheath complex. Usually (70%) unilateral, and painful. You will often see intracranial or spinal cord demyelination - in the setting of Devic's (neuromyelitis optica).

Papilledema: This is really an eye exam thing. Having said that you can sometimes see dilation of the optic nerve sheath.

Thyroid Orbitopathy: This is seen in 1/4 of the Graves cases and is the most common cause of exophthalmos. The antibodies that activate TSH receptors also activate orbital fibroblasts and adipocytes.

Things to know:

- Risk of compressive optic neuropathy
- Enlargement of **ONLY MUSCLE BELLY** (spares tendon) - different than *pseudo tumor*
- NOT Painful - different than *pseudo tumor*
- Order of Involvement:
IR > MR > SR > LR > SO "I'M SLOW"



Thyroid Orbit - *Spares Tendon Insertion*

Section 3: Spine

Anatomy Trivia

Cord Blood Supply: There is an anterior blood supply and a posterior blood supply to the cord. These guys get taken out with different clinical syndromes.

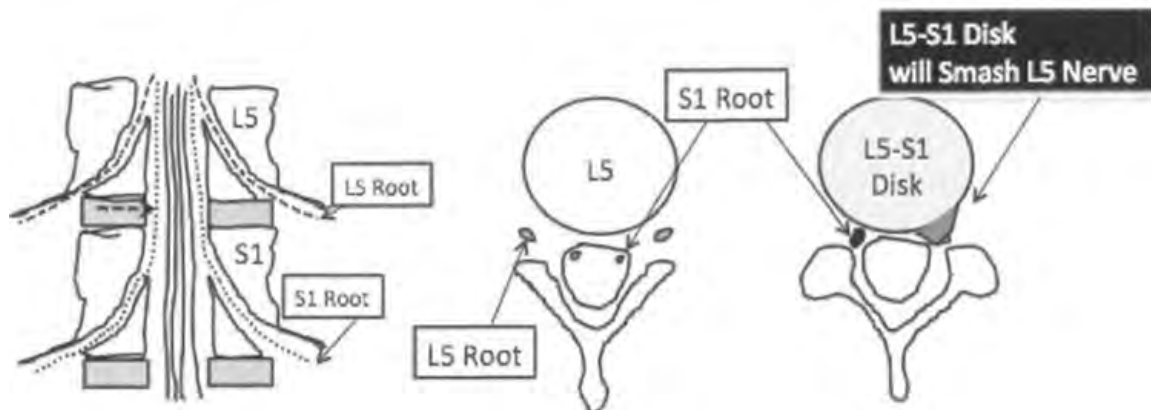
- *Anterior spinal artery* - arises bilaterally as two small branches at the level of the termination of the vertebral arteries. These two arteries join around the level of the foramen magnum.
- *Artery of Adamkiewicz* - This is the most notable reinforcer of the anterior spinal artery. In 75% of people it **comes off the left side of the aorta between T8 and T1**. It supplies the lower 2/3 of the cord. This thing can get covered with the placement of an endovascular stent graft for aneurysm or dissection repair leading to spine infarct.
- *Posterior Spinal Artery* - arises from either the vertebral arteries or the posterior inferior cerebellar artery. Unlike the anterior spinal artery this one is somewhat discontinuous and reinforced by multiple segmental or radiculopial branches.

Conus Medullaris: This is the terminal end of the spinal cord. It usually terminates at around L1. Below the inferior endplate of the L2 / L3 body should make you think tethered cord (especially if shown in a multiple choice setting).

Which nerve is compressed: There are 31 pairs of spinal nerves, with each pair corresponding to the adjacent vertebra - the notable exception being the “C8” nerve. Cervical disc herniations are less common than lumbar ones. The question is most likely to take place in the lumbar spine (the same spot most disc herniations occur). In fact more than 90% of herniations occur at L4-L5, and L5-S1.

You could see a question asking for nerve root compression in the canal, in the lateral recess, or in the NF.

- L5- S1 = Lateral Recess / Neural Foramina = L5
- L5-S1 = Central Canal = S1



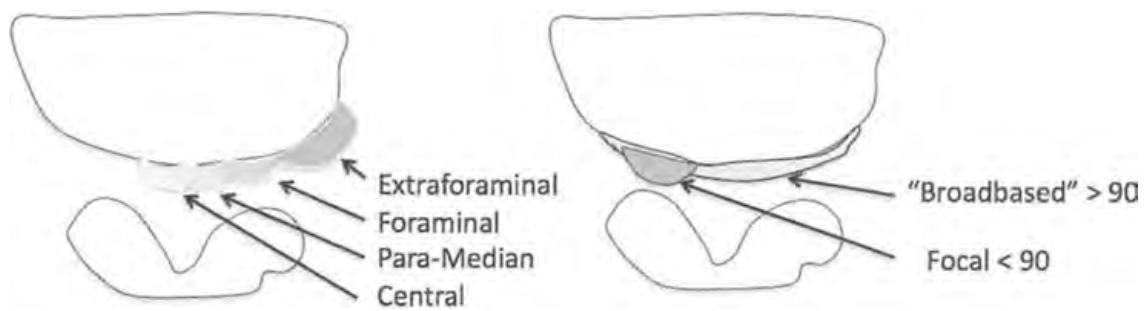
Epidural Fat: The epidural fat is not evenly distributed. The epidural space in the cervical cord is predominantly filled with venous plexus (as apposed to fat). In the lumbar spine there is fat both anterior and posterior to the cord.

Degenerative Changes

Stenosis: Spinal stenosis can be congenital (associated with short pedicles) or be acquired. The Torg-Pavlov ratio can be used to call it (vertebral body width to cervical canal diameter < 0.85). Symptomatic stenosis is more common in the cervical spine (versus the thoracic spine or lumbar spine). You can get some congenital stenosis in the lumbar spine from short pedicles, but it's generally not symptomatic until middle age.

Disc Nomenclature: In order to "improve accuracy" with regard to the lumbar spine various administrative regulatory bodies have decided on the vocabulary you are allowed to use.

- "Focal Herniation" is a herniated disc less comprising than 90 degrees of the disc circumference.
- "Broadbased Herniation" is a herniated disc in between 90-180 degrees.
- "Protrusion" - is a term used when the distance between the edge of the disc herniation is less than the distance between the edges of the base
- "Extrusion" is a term used when the edges of the disc are greater than the distance of the base.



Schmorl Node: This is a herniation of disc material through a defect in the vertebral body endplate into the actual marrow.

Scheuermann's - This is multiple levels (at least 3) of Schmorl's nodes in the spine of a teenager, resulting in kyphotic deformity (40 degrees in thoracic or 30 degrees in thoracolumbar).

Limbus Vertebra - This is a fracture mimic, that is the result of herniated disc material between the non-fused apophysis and adjacent vertebral body.

Endplate Changes: Commonly referred to as "Modic Changes." There is a progression in the MRI signal characteristics that makes sense if you think about it. You start out with degenerative changes causing irritation / inflammation so there is edema (T2 bright). This progresses to chronic inflammation with leads to some fatty change - just like in the bowel of an IBD patient - causing T1 bright signal. Finally, the whole thing gets burned out and fibrotic and it's T1 and T2 dark. As a prominent factoid, Type 1 changes look a lot like Osteomyelitis (clinical correlation in recommended).

Modic - Endplate Marrow Changes
Associated with Degenerative Disease

Type 1 "Edema"	T1 Dark, T2 Bright
Type 2 "Fat"	T1 Bright, T2 Bright
Type 3 "Scar"	T 1 Dark, T2 Dark

Annular Tears: You can have tears in your dorsal annulus. They are usually bright on T2 and have a curvilinear look. They may be a source of pain but are also seen as incidentals.

Myelogram Technique

Big point: Contrast should flow freely away from the needle tip gradually filling the thecal sac. The outlining of the the cauda equine is another promising sign that you did it right. If contrast pools at the needle tip or along the posterior or lateral thecal sac without free-flow, a subdural injection or injection in the fat around the thecal sac should be suspected.

Prior to the LP (*per A CR-ASNR recommendations*)

- STOP Coumadin 4-5 days
- STOP Plavix for 7 days
- Hold LMW Heparin for 12 hours
- Hold Heparin for 2-4 hours - document normal PTT
- Aspirin and NSAIDs are fine (not contraindicated)

Post Operative Back Trivia:

“Failed Back Surgery Syndrome” -Another entity invented by NEJM to take down the surgical subspecialties. These surgeons generally go from a non-indicated spine surgery, to a non-indicated leg amputation, to a non-indicated tonsillectomy on an innocent child. Text books will define it as recurrent or residual low back pain in the patient after disk surgery. This occurs about 40% of the time (probably more), since most back surgery is not indicated and done on inappropriate candidates. Causes of FBSS are grouped into early and late for the purpose of multiple choice test question writing:

Early FBSS	Late FBSS
Epidural Abscess	Epidural Fibrosis
	Recurrent Disk Herniation
	Arachnoiditis

Complications of Spine Surgery	
Recurrent Residual Disk	Will lack enhancement (unlike a scar - which will enhance on delays)
Epidural Fibrosis	Scar, that is usually posterior, and enhance homogeneously
Arachnoiditis	Buzzwords are “ <i>clumped nerve roots</i> ” and “ <i>empty thecal sac</i> ”, Enhancement for 6 weeks post op is considered normal. After 6 weeks may be infectious or inflammatory.
Conjoined Nerve Roots	Two adjacent nerve roots sharing an enlarged common sleeve - at a point during their exit from the thecal sac
12,000 Square Foot Mansion Syndrome	As spine surgeons perform more and more unnecessary surgeries they need something to spend all that money on.

Trauma to the Spine:

<i>Jefferson</i>	<i>Burst Fracture of C1</i>	<i>Axial Loading</i>
<i>Hangman</i>	<i>Bilateral Pedicle or Pars Fracture of C2</i>	<i>Hyperextension</i>
<i>Teardrop</i>	<i>Can be flexion or extension</i>	<i>Flexion (more common)</i>
<i>Clay-Shovelers</i>	<i>Avulsion of spinous process at C7 or T1</i>	<i>Hyperflexion</i>
<i>Chance</i>	<i>Horizontal Fracture through thoracolumbar spine ("seatbelt").</i>	

Jefferson Fracture: This is an axial loading injury (jumping into a shallow pool) - with the blow typically to the top of the head. The anterior and posterior arches blow out laterally. They would most likely show it on a plain film open mouth odontoid view (the CT would be too easy). Remember the C1 lateral masses shouldn't slide off laterally.

Important trivia to remember: **Neurologic (cord) damage is rare**, because all the force is directed into the bones.

Odontoid Fracture Classification:

- Type 1: Upper part of Odontoid (maybe stable) - this is actually rare
- Type 2: Fracture at the base - unstable
- Type 3: Fracture through dens into the body of C2. This is unstable, but has the best prognosis for healing.

Os Odontoideum: A mimic for a type 1 Odontoid fracture. This is an ossicle located at the position of the normal odontoid tip (the orthotopic position). The base of the dens is usually hypoplastic. The thing to know are that **this is prone to subluxation and instability**. It's association with Morquio's syndrome.

Orthotopic vs Dystopic: *Orthotopic is the position right on top of the dens. Dystopic is when it's fused to the clivus.*

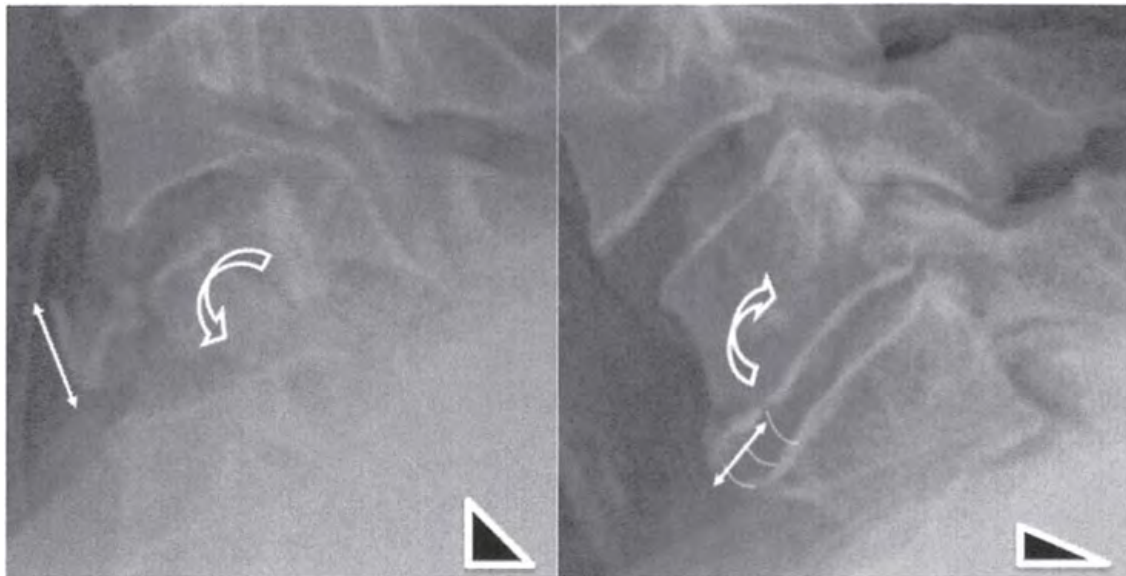
Hangman's Fracture: Seen most commonly when the chin hits the dashboard in an MVA ("direct blow to the face"). The **fracture is through the bilateral pars at C2** (or the pedicles - which is less likely). You will have anterior subluxation of the C2 body. Cord damage is actually uncommon with these, as the acquired pars defect allows for canal widening. There is often an associated fracture of the anterior inferior corner at C2 - from avulsion of the anterior longitudinal ligament.

Flexion Teardrop: This represents a teardrop shaped fracture fragment at the anterior-inferior vertebral body. Flexion injury is bad because it is associated with anterior cord syndrome (85% of patients have deficits). This is an unstable fracture, associated with posterior subluxation of the vertebral body.

Anterior Cord Syndrome: The anterior portion of the cord is jacked. Motor function and anterior column sensations (pain and temperature) are history. The dorsal column sensations (proprioception and vibration) are still intact.

Extension Teardrop: Another anterior inferior teardrop shaped fragment with avulsion of the anterior longitudinal ligament. This is less serious than the flexion type.

Flexion Teardrop	Extension Teardrop
Impaction Injury	Distraction Injury
Unstable	Stable (maybe)
Hyperflexion	Hyperextension
Classic History: “Ran into wall”	Classic History” Hit from behind”



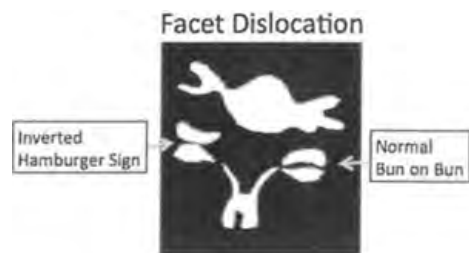
Flexion

Extension

Clay-Shoveler’s Fracture: This is an avulsion injury of a lower cervical / upper thoracic spinous process (usually C1). It is the result of a forceful hyperflexion movement (like shoveling). The “ghost sign” describes a double spinous process at C6-C7.

Facet Dislocation: This is a spectrum: Subluxed facets -> Perched -> Locked.

- **Unilateral:** If you have a unilateral facet (usually from hyperflexion and rotation) the superior facet slides over the inferior facet and gets locked. The unilateral is a stable injury. You will have the inverted hamburger sign on axial imaging on the dislocated side.



- **Bilateral:** This is the result of severe hyperflexion. You are going to have disruption of the posterior ligament complex. When this is full on you are going to have the dislocated vertebra displaced forward one -half the AP diameter of the vertebral body. This is highly unstable, and strongly associated with cord injury.

Atlantoaxial Instability - The articulation between C1 and C2 allows for lateral movement (shaking your head no). The transverse cruciform ligament straps the dens to the anterior arch of C1. The distance between the anterior arch and dens shouldn't be more than 5mm. The thing to know is the **association with Down syndrome and juvenile RA**. Rotary subluxation can occur in children without a fracture, with the kid stuck in a "cock-robbin" position - which looks like torticollis. Actually differentiating from torticollis is difficult and may require dynamic maneuvers on the scanner. This never, ever, ever happens in the absence of a fracture in an adult (who doesn't have Downs or RA). Having said that people over call this all the time in adults who have their heads turned in the scanner.

Benign Vs iMalignant Compression Fracture: This is a high yield distinction and practical in the real world.

Malignant	Benign
Convex posterior vertebral body cortex	Retropulsed fragment
Involves Posterior Elements	Transverse T1 and T2 dark band
Epidural / Paraspinal Mass	
Multiple Lesions	

Trauma to the Cord: There is a known correlation between spinal cord edema length and outcome. Having said that, you need to know the **most important factor for outcome is the presence of a hemorrhagic spinal cord injury** (these do very very badly).

Spinal Cord Syndromes		
Central Cord	Old lady with spondylosis or young person with bad extension injury.	Upper Extremity Deficit is worse than lower (corticospinal tracts are lateral in lower extremity)
Anterior Cord	Flexion Injury	Immediate Paralysis
Brown Sequard	Rotation injury or penetrating trauma	One half motor, other half sensory
Posterior Cord	Uncommon - but sometimes seen with hyperextension	Proprioception gone

Congenital /Developmental

Pars Interarticularis Defects: This is considered a fatigue or stress fracture, probably developing in childhood (they are more common in athletic kids). They are usually not symptomatic (only 25% are). The most common level is L5-S1 (90%), with all the rest at L4-L5. They tend to have more spondylolisthesis and associated degenerative change at L4-L5 than L5-S1. They can be seen on the oblique plain film as a “collar on the scottie dog.”

Pars Defects with Anteriorlithesis will have neuroforaminal stenosis, with spinal canal widening (when severe will have spinal canal stenosis as well).

Terminal Ventricle (ventricularis terminalis): This is a developmental variant. Normally, a large portion of the distal cord involutes in a late stage of spinal cord embryology. Sometimes this process is not uniform and you get stuck with a stupid looking cyst at the end of your cord. These things are usually small (around 4mm), and cause no symptoms. Sometimes they can get very big (like this example) and cause some neurologic symptoms.



Spinal Dysraphism:

You can group these as open or closed (closed with and without a mass). Open means neural tissue exposed through a defect in bone and skin (spina bifida aperta). Closed means the defect is covered by skin (spina bifida occulta).

Open Spinal Dysraphisms: This is the result of a failure of the closure of the primary neural tube, with obvious exposure of the neural placode through a midline defect of the skin. You have a dorsal defect in the posterior elements. The **cord is going to be tethered**. There is an association with diastematomyelia and Chiari II malformations. Early surgery is the treatment / standard of care.

- *Myeloceles:* This is the more rare type where the neural placode is flush with the skin.
- *Myelomeningoceles:* This is the more common type (98%) where the neural placode protrudes above the skin. These are more common with Chiari II malformations.

Closed Spinal Dysraphisms with Subcutaneous Mass

- *Meningoceles:* This is herniation of a CSF filled sac through a defect in the posterior elements (spina bifida). It is most typical in the lumbar or sacral periods. Although they can occur in the cervical spine. They may be anterior (usually pre-sacral). An important point is **that neural tissue is not present in the sac**.
- *Lipomyelocele /Lipomyelomeningoceles:* These are lipomas with a dural defect. On exam you are going to have a subcutaneous fatty mass above the gluteal crease. **These are 100% associated with tethered cord** (myelomeningocele may or may not).
- *Terminal Myelocystocele* - This is a herniation of the terminal syrinx into a posterior meningocele via a posterior spinal defect.

Closed Spinal Dysraphisms without Subcutaneous Mass

- *Intradural lipomas* - Most common in the thoracic spine also the dorsal aspect. They don't need to be (but can be) associated with posterior element defects.
- *Fibrolipoma of the filum terminale* - This is often an incidental finding. There will be a linear T1 bright structure in the filum terminale. The filum is not going to be unusually thickened and the conus will be normally located.

- *Tightfilum terminale* - This is a thickened filum terminale (> 2mm), with a low lying conus (below the inferior endplate of L2). You may have an associated terminal lipoma. The “**tethered cord syndrome**” is based on the clinical findings of low back pain and leg pain plus urinary bladder dysfunction. This is the result of stretching the cord with growth of the canal.
- *Dermal Sinus* - This is an epithelium lined tract that extends from the skin to deep soft tissues (sometimes the spinal canal, sometimes a dermoid or lipoma). These are T₁ low signal (relative to the background high signal from fat).

Diastematomyelia - This describes a sagittal split in the spinal cord. They almost always occur between T9-S1, with normal cord both above and below the split. You can have two thecal sacs (or just one), and each hemicord has its own central canal and dorsal/ventral horns. Classification systems are based on the presence / absence of an osseous or fibrous spur and duplication or non-duplication of the thecal sac.

Caudal Regression: This is a spectrum of defect in the caudal region that ranges from partial agenesis of the coccyx to lumbosacral agenesis. The associations to know are VACTERL and Currarino triad. Think about this with maternal diabetes. “Blunted sharp” high terminating cord is classic, with a “shield sign” from the opposed iliac bones (no sacrum).

Currarino Triad:

*Anterior Sacral Meningocele,
Anorectal malformation,
Sacroccygeal osseous defect
(similar sacrum).*

Spinal Vascular Disorders

AVFs/AVMs: There are 4 types. **Type 1 is by far the most common (85%). It is a Dural AVF;** the result of a fistula between the dorsal radiculomedullary arteries and radiculomedullary vein / coronal sinus - with the dural nerve sleeve. It is acquired and seen in older patients who present with progressive radiculomyelopathy. The most common location is the thoracic spine. If anyone asks the “gold standard for diagnosis is angiography” , although CTA or MRA will get the job done. You will have T2 high signal in the central cord (which will be swollen), with serpentine perimedullary flow voids (which are usually dorsal).

<i>Spinal AVM/AVFs</i>	
Type 1	Most Common type (85%). Dural AVF - with a single coiled vessel
Type 2	Intramedullary Nidus from anterior spinal artery or posterior spinal artery. Can have aneurysms, and can bleed. Most common presentation is SAH. Associated with HHT and KTS (other vascular syndromes).
Type 3	Juvenile, very rare, often complex and with a terrible prognosis
Type 4	Intradural perimedullary with subtypes depending on single vs multiple arterial supply. These tend to occur near the conus.

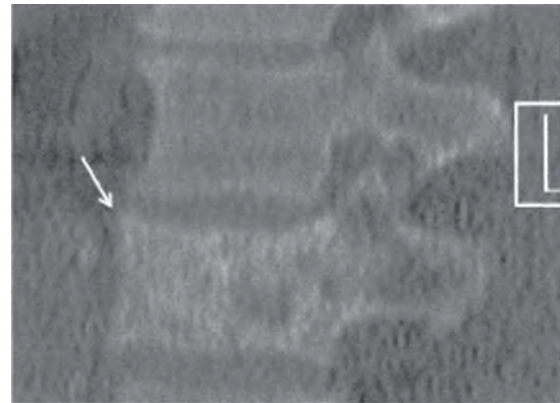
Foix Alajouanine Syndrome: This is a myelopathy association with a Dural AVF. The classic history is a 45 year old male with lower extremity weakness and sensory deficits. You have increased T2 signal (either at the conus, or lower thoracic spine), with associated prominent vessels. The underlying pathophysiology is venous hypertension.

Misc Disorders Affecting the Spine

Pagets - This is discussed in detail in the MSK chapter, but is such a high yield topic that it's worth touching on again. The incidence increases with age (around 8% at age 80). It's at increased risk for fracture, and has a 1% risk of sarcoma degeneration (usually high grade).

It's shown two ways in the spine:

- (1) An enlarged "ivory vertebrae",
- (2) Picture frame vertebrae (sclerotic border).



Ivory Vertebrae - Pagets (*ormets*)

Renal Osteodystrophy - Another high yield topic covered in depth in the MSK chapter. The way it's shown in the spine is the "Rugger Jersey Spine" - with sclerotic bands at the top and bottom of the vertebral body. You could also have paraspinal soft tissue calcifications

Osteopetrosis - Another high yield topic covered in depth in the MSK chapter. This is a genetic disease with impaired osteoclastic resorption. You have thick cortical bone, with diminished marrow. On plain film or CT it can look like a Rugger Jersey Spine or Sandwich vertebra. On MR you are going to have loss of the normal T1 bright marrow signal, so it will be T1 and T2 dark.

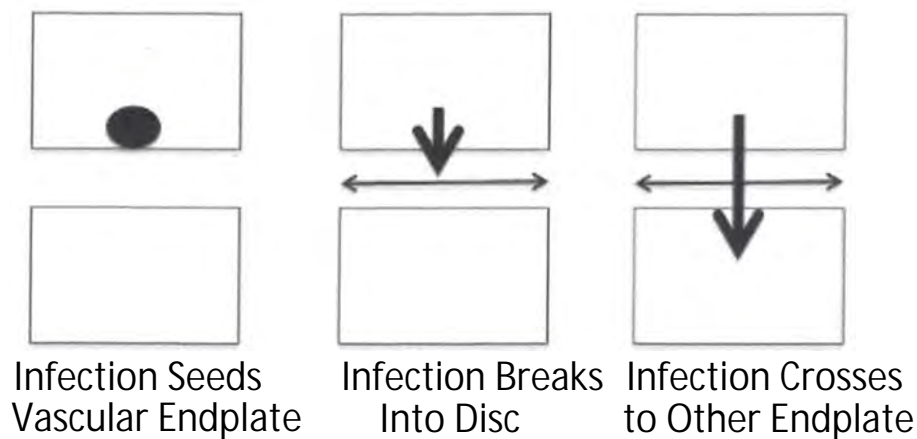
"H-Shaped Vertebra" - This is usually a **buzzword for sickle cell**, although it's only seen in about 10% of cases. It results from microvascular endplate infarct. If you see "H-Shaped vertebra" the answer is sickle cell. If sickle cell isn't a choice the answer is Gauchers.

Another tricky way to ask this is to say which of the following causes **"widening of the disc space."** Widened disc space is another way of describing a "H Shape" without saying that.

Infectious

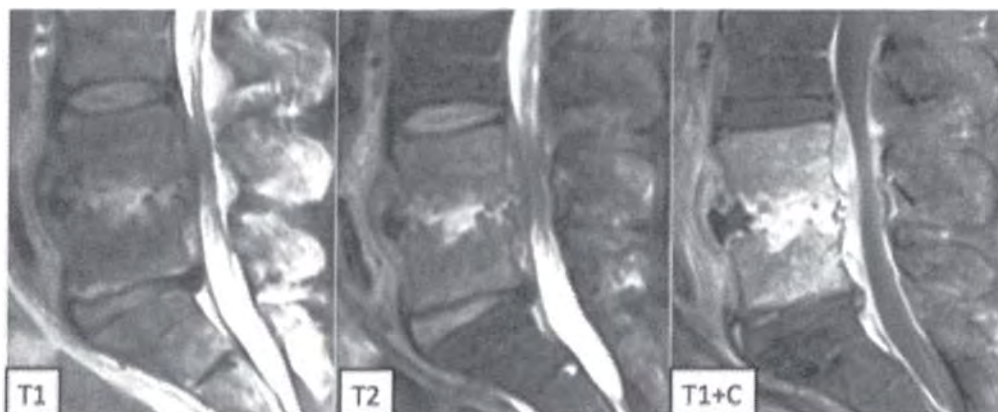
Discitis / Osteomyelitis:

Infection of the disc and infection of the vertebral body nearly always go together. The reason has to do with the route of seeding; which typically involves seeding of the vertebral endplate (which is vascular), subsequent eruption and crossing into the disc space, and eventual involvement of the adjacent vertebral body.



In adults, the source is usually from a recent surgery, procedure, or systemic infection. In children it's usually from hematogenous spread. For the Step 1 trivia: Staph A is the most common bug, and always think about an IV drug user. Almost always (80%) of the time the ESR and CRP are elevated.

Imaging: Early on it's very hard to see with plain films, you will need MRI. You are looking for paraspinal and epidural inflammation, T2 bright disc signal, disc enhancement



Vertebral Osteomyelitis

Pott's Disease: TB of the spine is more common in “developing” countries. It behaves in a few different ways, and that makes it easy to test on.

Things to know about TB in the spine:

- *It tends to **spare the disc space***
- *It tends to have multi-level thoracic “skip” involvement*
- *Buzzword “Calcified Psoas Abscess ”*
- *Buzzword “Gibbus Deformity ” - which is a destructive focal kyphosis*

Brucellosis: This is uncommon in the USA. It's somewhat unique feature make it testable. Just know that it **favours the lower lumbar spine and SI joints**. Vertebral destruction and paraspinal abscess are actually less common. Step 1 buzzword was “cow's milk” or “farm exposure.”

Epidural Abscess: This is an infected collection between the dura and periosteum. Its usually MRSA and the patient usually has HIV or is a bad diabetic. This is most likely to be shown with diffusion. A collection that restricts is going to be an abscess (in the spine and in the brain) - most of the time. It should also be T1 dark, T2 bright, with peripheral enhancement.

Cord Pathology

Syrinx - This is a term that means the same thing as hydromyelia, syringomyelia, hydrosyringomyelia, syringohydromyelia, and syringobulbia - at least to Radiologists. Syring is easier to say than all those other terms, and therefore more commonly used. Most (90%) are congenital, and associated with Chiari I and II, as well as Dandy-Walker, Klippel-Feil, and Myelomeningoceles. The other 10% are acquired either by trauma, tumor, or vascular insufficiency.

Demyelinating:

Broadly you can think of cord pathology in 5 categories: Demyelinating, Tumor, Vascular, Inflammatory, and Infectious.

In the real world, the answer is almost always MS - which is by far the most common cause. The other three things it could be are Neuromyelitis Optica (NMO), acute disseminating encephalomyelitis (ADEM) or Transverse Myelitis (TM).

MS	Usually Short Segment	Usually Part of the Cord	Not swollen, or Less Swollen	Can Enhance/ Restrict when Acute
TM	Usually Long Segment	Usually involves both sides of the cord	Expanded Swollen Cord	Can Enhance
NMO	Usually Long Segment	Usually involves both sides of the cord		Optic Nerves Involved
ADEM			Not swollen, or Less Swollen	
Infarct	Usually Long Segment			Restricted Diffusion
Tumor			Expanded Swollen Cord	Can Enhance

MS in the Cord: "Multiple lesions, over space and time." The lesions in the spine are typically short segment (< 2), usually only affect half / part of the cord. The cervical cord is the most common location. There are usually lesions in the brain, if you have lesions in the cord (isolated cord lesions occur about 10% of the time). The lesions can enhance when acute - but this is more common than in the brain. You can sometimes see cord atrophy if the lesion burden is large.

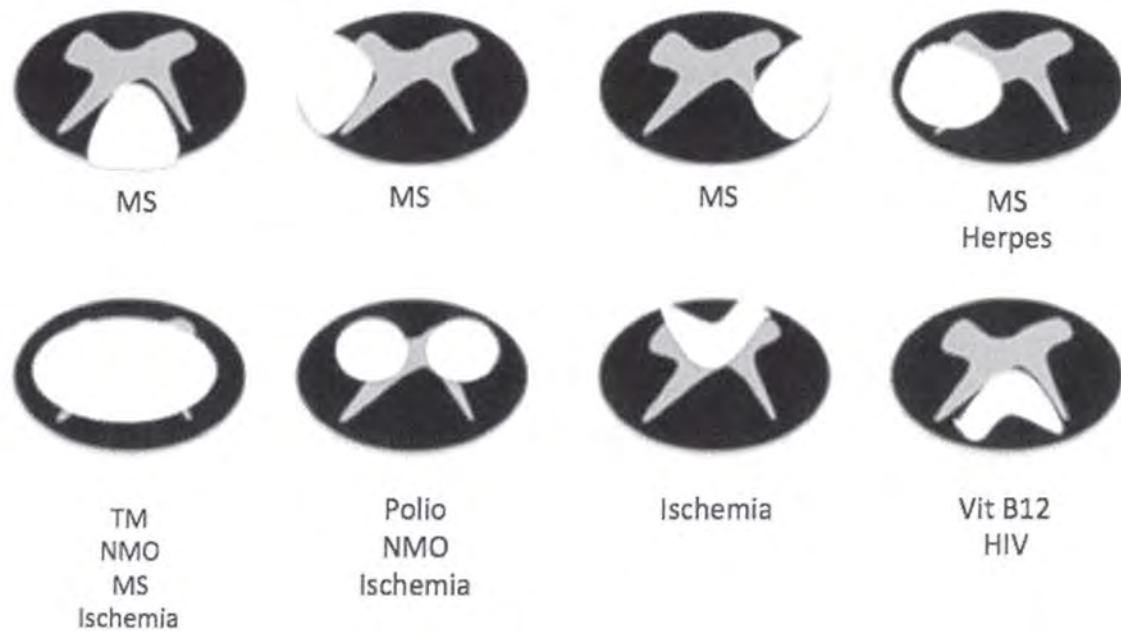
Transverse Myelitis: This is a focal inflammation of the cord. The causes are numerous (infectious, post vaccination - classic rabies, SLE, Sjogrens, Paraneoplastic, AV-malformations). You typically have at least 2/3 of the cross sectional area of the cord involved, and focal enlargement of the cord. Spliters with use the terms “Acute partial” for lesions less than two segments, and “acute complete” for lesions more than two segments. The factoid to know is that the “Acute partials” are higher risk for developing MS.

ADEM: As described in the brain section, this is usually seen after a viral illness or infection typically in a child or young adult. The lesions favor the dorsal white matter (but can involve grey matter). As a pearl, the presence of cranial nerve enhancement is suggestive of ADEM. The step 1 trivia, is that the “anti-MOG IgG” test is positive in 50% of cases. Just like MS there are usually brain lesions (although ADEM lesions can occur in the basal ganglia and pons - which is unusual in MS).

NMO (Neuromyelitis Optica): This is also sometimes called Devics. It can be monophasic or relapsing, and favors the optic nerves and cervical cord. Tends to be longer segment than MS, and involve the full transverse diameter of the cord. Brain lesions can occur (more commonly in Asians) and are usually periventricular. If any PhDs ask the reason the periventricular location occurs is that the antibody (NMO IgG) attacks the Aquaporin 4 channels - which are found in highest concentration around the ventricles.

Subacute Combined Degeneration: This is a fancy way of describing the effects of a Vitamin B12 deficiency. The classic look is **bilateral symmetrically increased T2 signal in the dorsal columns**, without enhancement. The appearance has been described as an “inverted V sign.” The signal change typically begins in the upper thoracic region with ascending or descending progression.

HIV Vacuolar Myelopathy: This has been described as a late finding in AIDS patients. You have spinal cord atrophy, and T2 high signal symmetrically involving the posterior columns. It looks like subacute combined degeneration.



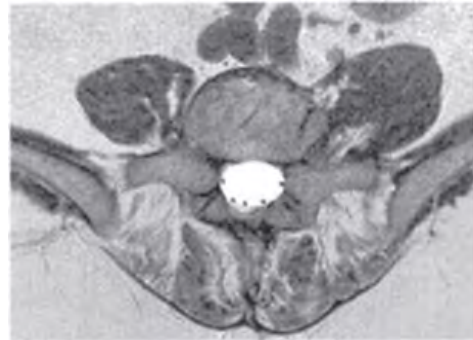
Spinal Cord Infarct: Cord infarct /ischemia can have a variety of causes. The most common cause is “idiopathic,” although I’d expect the most common multiple choice scenario to revolve around treating an aneurysm with a stent graft, or embolizing a bronchial artery. Impairment involving the anterior spinal artery distribution is most common. With anterior spinal artery involvement you are going to have central cord / anterior horn cell high signal on T2 (because gray matter is more vulnerable to ischemia). The **“owl eye appearance”** of anterior spinal cord infarct is a buzzword. It’s usually a long segment, being more than 2 segments. Diffusion using single shot fast spin echo or line scan can be used with high sensitivity (to compensate for artifacts from spinal fluid movement).

Inflammatory / Infectious:

Arachnoiditis: This is a general term for inflammation of the subarachnoid space. It can be infectious but can also be post-surgical. **It actually occurs about 10-15% of the time after spine surgery, and can be a source of persistent pain / failed back.**

It's shown two ways:

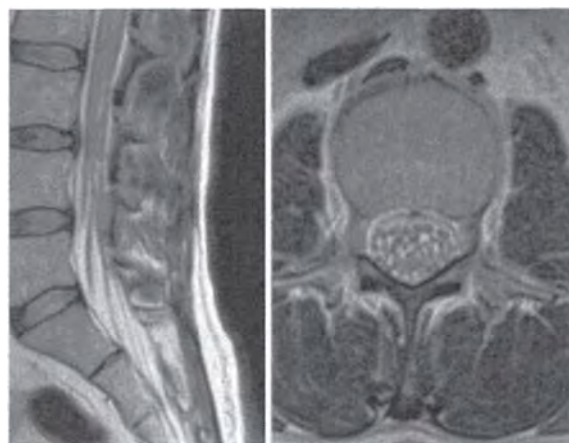
- (1) **Empty Thecal Sac Sign** - Nerve roots are adherent peripherally, giving the appearance of an empty sac.
- (2) Central Nerve Root Clumping. This can range in severity from a few nerves clumping together, to all of them fused into a single central scarred band.



Empty Thecal Sac

Guillain Barre Syndrome (GBS) - One of those weird auto-immune disorders that causes ascending flaccid paralysis. The step 1 trivia was **Campylobacter**, but you can also see it after surgery, or in patients with lymphoma or SLE. The thing to know is **enhancement of the nerve roots of the cauda equina**. Other pieces of trivia that are less likely to be asked are that the facial nerve is the most common cranial nerve affected, and that the anterior spinal roots enhance more than the posterior ones.

Chronic Inflammatory Demyelinating Polyneuropathy (CIDP) - The chronic counterpart to GBS. Clinically this has a gradual and protracted weakness (GBS improves in 8 weeks, CIDP does not). The buzzword is thickened, enhancing, "onion bulb" nerve roots.

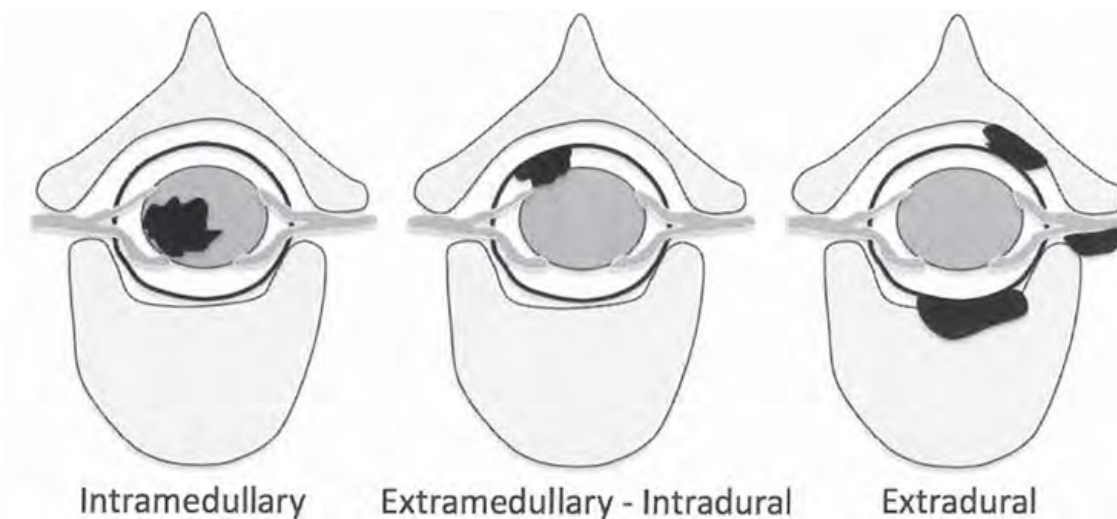


CIDP - Diffuse Thickening of the Nerve Roots

Tumor:

The classic teaching is to first describe the location of the tumor, as either (1) Intramedullary, (2) Extramedullary Intradural, or (3) Extradural. This is often easier said than done.

Differentials are based on the location.



Intramedullary	Astrocytoma, Ependymoma, Hemangioblastoma
Extramedullary Intradural	Schwannoma, Meningioma, Neurofibroma, Drop Mets
Extradural	Disc Disease (most common), Bone Tumors, Mets, Lymphoma

Intramedullary:

- **Astrocytoma** - This is the most common intramedullary tumor in peds. It favors the upper thoracic spine. There will be fusiform dilation of the cord over multiple segments. They are dark on T1, bright on T2, and they enhance. They may be associated with rostral or caudal cysts which are usually benign syrinx formation.

Astrocytoma	Ependymoma
More in Cervical Cord	More in Lower Cord
Eccentric	Central
	More Often Hemorrhagic

- **Ependymoma** - This is the most common primary cord tumor of the lower spinal cord, conus / filum terminale. This is the **most common intramedullary mass in adults**. Although you can certainly see them in the cervical cord as well. The **“myxopapillary form”** is exclusively found in the conus / filum locations. They can be hemorrhagic, and have a **dark cap on T2**. They have tumoral cysts about 1/4 of the time. They are a typically long segment (averaging 4 segments).



Ependymoma

- **Hemangioblastoma** - These are associated with Von Hippel Lindau (30%). The thoracic level is favored (second most common is cervical). The classic look is a wide cord, with considerable edema. Adjacent serpiginous draining meningeal varicosities can be seen.
- **Intramedullary Mets:** This is very very rare, but when it does happen it's usually lung (70%).

VHL Associations:

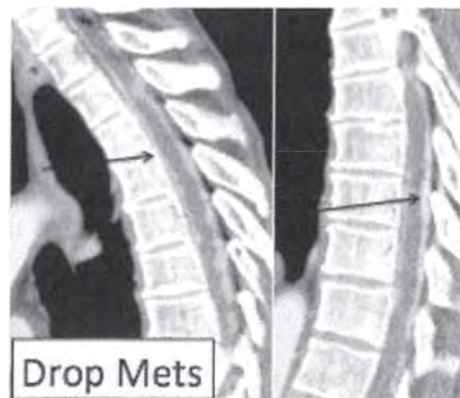
- Pheochromocytoma
- CNS Hemangioblastoma (cerebellum 75%, spine 25%)
- Endolymphatic Sac Tumor
- Pancreatic Cysts
- Pancreatic Islet Cell Tumors
- Clear Cell RCC

Extramedullary Intradural:

- **Schwannoma:** This is the most common tumor to occur in the Extramedullary Intradural location. They are benign, usually solitary, usually arising from the dorsal nerve roots. This can be multiple in the setting of NF-2 and the Carney Complex. The appearance is variable, but the classic look is a dumbbell with the skinny handle being the intraforaminal component. They are T1 dark, T2 bright, and will enhance. They look a lot like neurofibromas. If they have central necrosis or hemorrhage that favors a schwannoma.
- **Neurofibroma:** This is another benign nerve tumor (*composed of all parts of the nerve: nerve + sheath*), that is also usually solitary. There are two flavors: solitary and plexiform. The plexiform is a multilevel bulky nerve enlargement that is pathognomonic for NF-1. Their lifetime risk for malignant degeneration is around 5-10%. Think about malignant degeneration in the setting of rapid growth. They look a lot like schwannomas. If they have a hyperintense T2 rim with a central area of low signal - “target sign” that makes you favor neurofibroma.

Schwannoma	Neurofibroma
Do NOT envelop the adjacent nerve root	Do envelop the adjacent nerve root (usually a dorsal sensory root)
Solitary	Solitary
Multiple makes you think NF-2	Associated with NF -1 (even when single)
Cystic change / Hemorrhage	T2 bright rim, T2 dark center “target sign”

- **Meningioma:** These guys adhere to but do not originate from the dura. They are more common in women (70%). They favor the posterior lateral thoracic spine, and the anterior cervical spine. They enhance brightly and homogeneously. They are often T1 iso to hypo, and slightly T2 bright. They can have calcifications.
- **Drop Mets:** Medulloblastoma is the most common primary tumor to drop. Breast cancer is the most common systemic tumor to drop (followed by lung and melanoma). The cancer may coat the cord or nerve root, leading to a fine layer of enhancement.

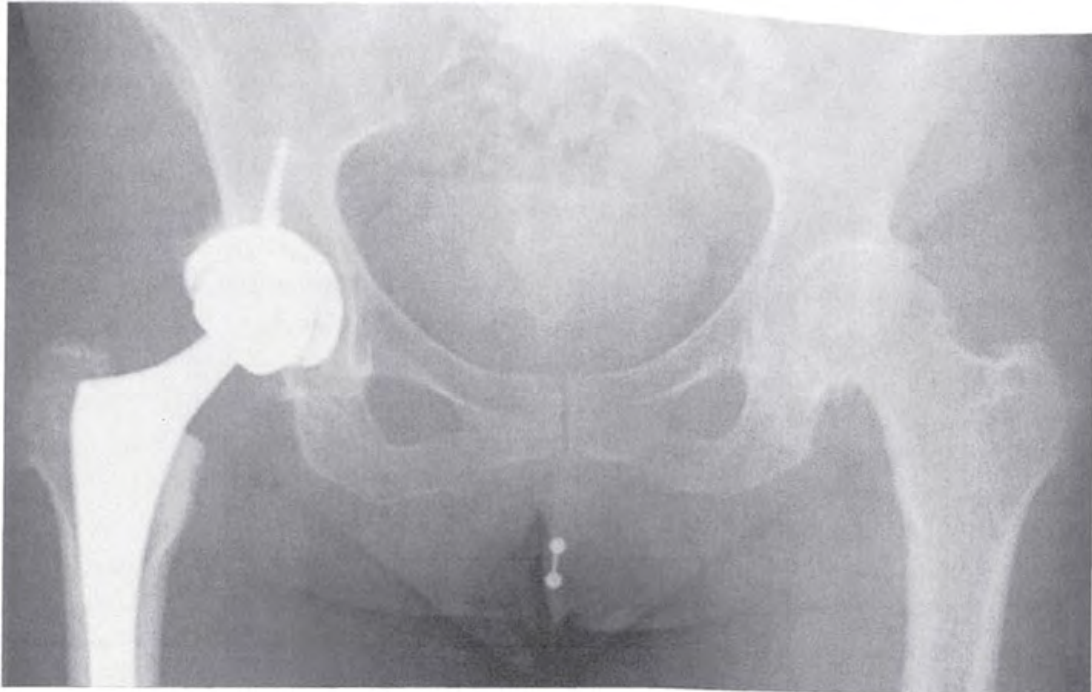


Extradural:

- **Vertebral Hemangioma:** These are very common - seen in about 10% of the population. They classically have thickening trabeculae appearing as parallel lineal densities “jail bar” or “corduroy” appearance. In the vertebral body they are T₁ and T₂ bright, although the extraosseous components typically lack fat and are isointense on T₁.
- **Osteoid Osteoma:** This is also covered in the MSK chapter, but as a brief review focusing on the spine they love to involve the posterior elements (75%), and are rare after age 30. They tend to have a nidus, and surrounding sclerosis. The nidus is T₂ bright, and will enhance. The classic story is night pain, better with aspirin. Radiofrequency ablation can treat them (under certain conditions).
- **Osteoblastoma:** This is similar to an Osteoid osteoma but larger than 1.5cm. Again, very often in the posterior elements - usually of the cervical spine.
- **Aneurysmal Bone Cyst:** These guys are also covered in the MSK chapter. They also like the posterior elements and are usually seen in the first two decades of life. They are expansile (as the name implies) and can have multiple fluid levels on T₂. They can get big, and look aggressive.
- **Giant Cell Tumor:** These guys are also covered in the MSK chapter. These are common in the sacrum, although rare anywhere else in the spine. You don't see them in young kids. If they show this it's going to be a **lytic expansile lesion in the sacrum with no rim of sclerosis**.
- **Vertebral Plana** - The pancake flat vertebral body. Just say Eosinophilic Granuloma in a kid (could be neuroblastoma met), and Mets / Myeloma in an adult.
- **Chordoma:** This is **most common in the sacrum** (they will want you to say clivus - that is actually number 2). The thing to know is that vertebral primary tend to be more aggressive / malignant than their counter parts in the clivus and sacrum. The classic story in the vertebral column is “involvement of two or more adjacent vertebral bodies with the intervening disc.” Most are **very T₂ bright**.
- **Leukemia:** They love to show it in the spine. You have loss of the normal fatty marrow - so it's going to be homogeneously dark on T₁. More on this in the MSK chapter. •
- **Mets:** The classic offenders are prostate, breast, lung, lymphoma, and myeloma. Think **multiple lesions, with low T₁ signal**. Cortical breakthrough or adjacent paravertebral components are also helpful.

12 Musculoskeletal

Prometheus Lionhart, M.D.



In the real world MSK is full of differentials. However, for multiple choice test taking differential cases make terrible questions. So the test writers are left with three options:
(1) Show a case with a differential but list 3 terrible distractors (these will be very easy),
(2) Show Aunt Minnies, or (3) Ask Trivia. As you read this chapter, I want you to focus on the testable trivia as this will make up the bulk of the more difficult questions.

High Yield Topics Include:

- Anatomy
- Arthritis
- Cystic Bone Lesions
- Pagets* - *Extremely High Yield*
- Hyperparathyroidism
- Pathology Shown With Ultrasound

Trauma /Acquired

Most third year radiology residents are excellent at basic MSK trauma. Certainly the purpose of this section is not to teach you how to read trauma x-rays. Instead the focus is on random trivia you might not know, that lends itself well to multiple choice test.

Basic Fracture Trivia:

- * *Stress fracture* is abnormal stress on normal bone.
- * An *insufficiency fracture* is normal stress on abnormal bone.
- * Bones heal in about 6-8 weeks (months for tibia), and remember that the osteolytic phase precedes new bone formation.

Hand/Wrist:

Scaphoid Fracture:

- * Most common carpal bone fracture
- * 70% at the waist
- * Blood supply is distal to proximal; with the proximal pole most susceptible to AVN (first sign of AVN is sclerosis)
- * Proximal fractures are most susceptible to AVN and non-union
- * Avulsion fractures occur at distal pole

SNAC Wrist (Scaphoid Non-Union Advanced Collapse)

- * The proximal scaphoid fragment usually remains attached to the lunate while the distal scaphoid fragment rotates into flexion. The result of this is abnormal contact in the **radioscaphoid compartment (arthritis there is the first sign)**
- * Eventually you will have the capitate migrating proximally, radially, and dorsal to the lunate (**DISI configuration**)

SLAC Wrist (Scapholunate Advanced Collapse)

- * Pattern of wrist gone bad, which can be post traumatic (*untreated scapholunate dissociation or chronic scaphoid non-union*) or related to arthritis (CPPD)
- * The most likely multiple choice question = **radioscaphoid joint is first to develop degenerative changes**
- * Capitate will migrate proximally and there will eventually be a **DISI deformity**
- * Treatments (based off patient's occupation and needs):
 - o Wrist Fusion = Maximum Strength, Loss of Motion
 - o Proximal Row Carpectomy = Maintain ROM, Lose Strength

Carpal Dislocations

- * This can be thought of as a spectrum of perilunate instability related to ligamentous injury:

- o Scapholunate Dissociation*

- Scapholunate ligament disrupted - widens the interval to 3mm

- “Terry Thomas Sign”

- Worse with clenched fist

- Chronic SL dissociation leads to SLAC wrist

- o Perilunate Dislocation*

- 60% associated with Scaphoid Fractures

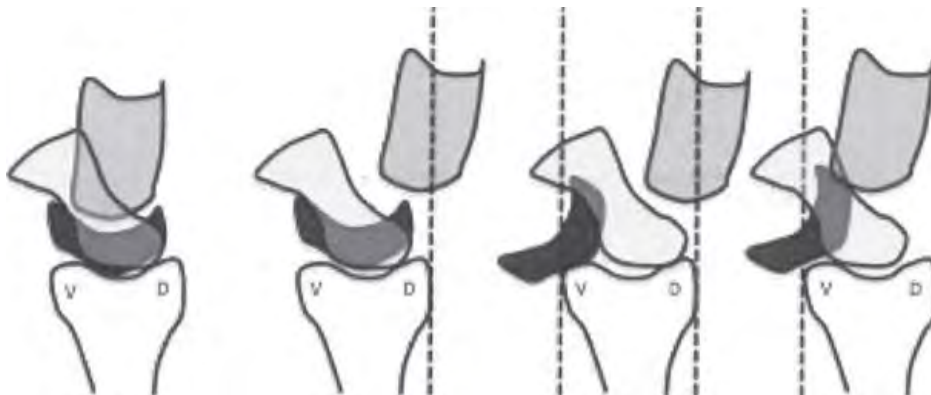
- o Midcarpal Dislocation*

- Triquetrolunate interosseous ligament disruption OR

- Triquetral Fracture

- o Lunate Dislocation -*

- Dorsal radiolunate ligament injury



Normal Perilunate Midcarpal Lunate

Dislocation Dislocation Dislocation

DISI vs VISI

- * Confusing and therefore high yield topic.
- * For practical purposes **VISI deformity almost never happens**, because you have to wreck your wrist so bad it is ridiculous (requires a lot of ligaments to go out). DISI deformity happens all the time. If the question is asking “what is associated?” the answer is probably DISI.
- * The way I remember them is that a “V” looks like an acute angle, so “V”ISI is the one with the scapholunate angle less than 30 (and the other one is the other one).

<i>This vs That:</i> DISI VS VISI	
DISI	VISI
SL Angle > 60 Degrees	SL Angle < 30 Degrees
CL Angle >30 Degrees	CL Angle > 30 Degrees
Radial Side Ligament Injury	Ulnar Side Ligament Injury

Bennett and Rolando Fractures

- * They are both fractures at the base of the first metacarpal
- * The Rolando fracture is comminuted
- * The multiple choice question is likely to be: The pull of the **Abductor pollicis longus** tendon is what causes the dorsolateral dislocation in the Bennett Fracture

Gamekeeper's Thumb

- * Avulsion fracture at the base of the proximal phalanx associated with **ulnar collateral ligament** disruption.
- * The frequently tested association is that of a "**Stener Lesion.**" A Stener Lesion is when the Adductor tendon gets caught in the torn edges of the UCL. The displaced ligament won't heal right, and will need surgery.

Carpal Tunnel Syndrome (CTS)

- * Median Nerve Distribution (thumb-radial aspect of 4th digit), often bilateral, and may have thenar muscle atrophy.
- * The question is most likely going to be "what goes through the tunnel?", and "what doesn't ?" - (see anatomy discussion at the end of section).
- * On Ultrasound, enlargement of the nerve is the main thing to look for
- * It's usually from repetitive trauma, but the **association with dialysis** is more likely to be tested.

Guyon's Canal Syndrome

- * Entrapment of the ulnar nerve as it passes through Guyon's canal (formed by the pisiform and the hamate - and the crap that connects them). Classically caused by handle bars "*handle bar palsy.*" Fracture of the hook of the hamate can also eat on that ulnar nerve.

Elbow / Forearm:

- * Radial Head Fracture is most common in adults (supracondylar is most common in PEDs)
 - o Sail sign (posterior is positive)
- * Capitellum fractures are associated with posterior dislocation

Eponyms:

- * *Essex-Lopresti*: Fracture of the radial head + Anterior dislocation of the distal radial ulnar joint
- * *Monteggia Fracture (MUGR)*: Fracture of the proximal ulna, with anterior dislocation of the radial head.
- * *Galeazzi Fracture (MUGR)*: Radial shaft fracture, with anterior dislocation of the ulna at the DRUJ.

The Dreaded PEDs elbow (*Covered in more detail in the Peds chapter*):

- * Anterior humeral line: through proximal 1/3 capitellum = supracondylar fracture
- * Radio-capitellar line: must always align, otherwise dislocated radial head
- * CRITOE: The avulsion of the medial epicondyle mimicking trochlear ossification is the frequent trick
- * *Nursemaid Elbow*: subluxation of the radial head (reduces with supination), X-ray is usually normal.

Cubital Tunnel Syndrome

- * The result of repetitive valgus stress
- * *Anatomy Trivia*: the site where the ulnar nerve passes beneath the cubital tunnel retinaculum also known as the epicondylo-olecranon ligament or Osborne band
- * Can occur from compression by any pathology (tumor, hematoma, etc...), when it occurs from an **accessory muscle** it's classically the **anconeus epitrochlearis**

Shoulder (*MR1 covered separately*)

Dislocation:

- * *Anterior inferior* (subcoracoid) are by far the most common (like 90%).
 - o Hill-Sachs is on the Humerus.
 - o Hill-Sachs is on the posterior lateral humerus, and *best seen on internal rotation view*.
 - o Bankart - anterior inferior labrum
 - o Greater tuberosity avulsion fracture occurs in 10-15% of anterior dislocations in patient's over 40.
- * *Posterior Dislocation*: uncommon - probably from seizure or electrocution
 - o *Rim Sign* - no overlap glenoid and humeral head
 - o *Trough Sign* - reverse Hill Sachs, impaction on anterior humerus
 - o Arm may be locked in internal rotation on all views
- * *Inferior Dislocation (luxatio erecta humeri)* - this is an uncommon form, where the arm is sticking straight over the head. The thing to know is 60% get neurologic injury (usually the axillary nerve).

Hill Sacs	Posterolateral humeral head impaction fracture (anterior dislocation)
Bankart	Anterior Glenoid Rim (anterior dislocation)
Trough Sign	Anterior humeral head impaction fracture (posterior dislocation)
Reverse Bankart	Posterior Glenoid Rim (posterior dislocation)

Memory Tool (works for me anyway)

I remember that hip dislocations are posterior - from the straight leg dashboard mechanism. Then I just remember that shoulders are the opposite of that (the other one, is the other one). **Shoulder = Usually Anterior**

Proximal Humerus Fracture: This is usually in an old lady falling on an out stretched arm. Orthopods use the Neer classifications (how many parts the humerus is in ?). Three or four part fractures tend to do worse.

Hip / Femur

Femoral Neck Fractures:

- * On the inside (**medial**) is the classic **stress** fracture location
- On the outside (**lateral**) is the classic **bisphosphonate** related fx location



Bisphosphonate Fracture (Lateral Femur)
***Stress would be medial*

Hip Fracture /Dislocation: You see these with dash board injuries. The **posterior dislocation** (almost always associated with a fracture as it's driven backwards) is much more common than the anterior dislocation.

Anterior Column vs Posterior Column - the acetabulum is supported by two columns of bone that merge together to form an “inverted Y”

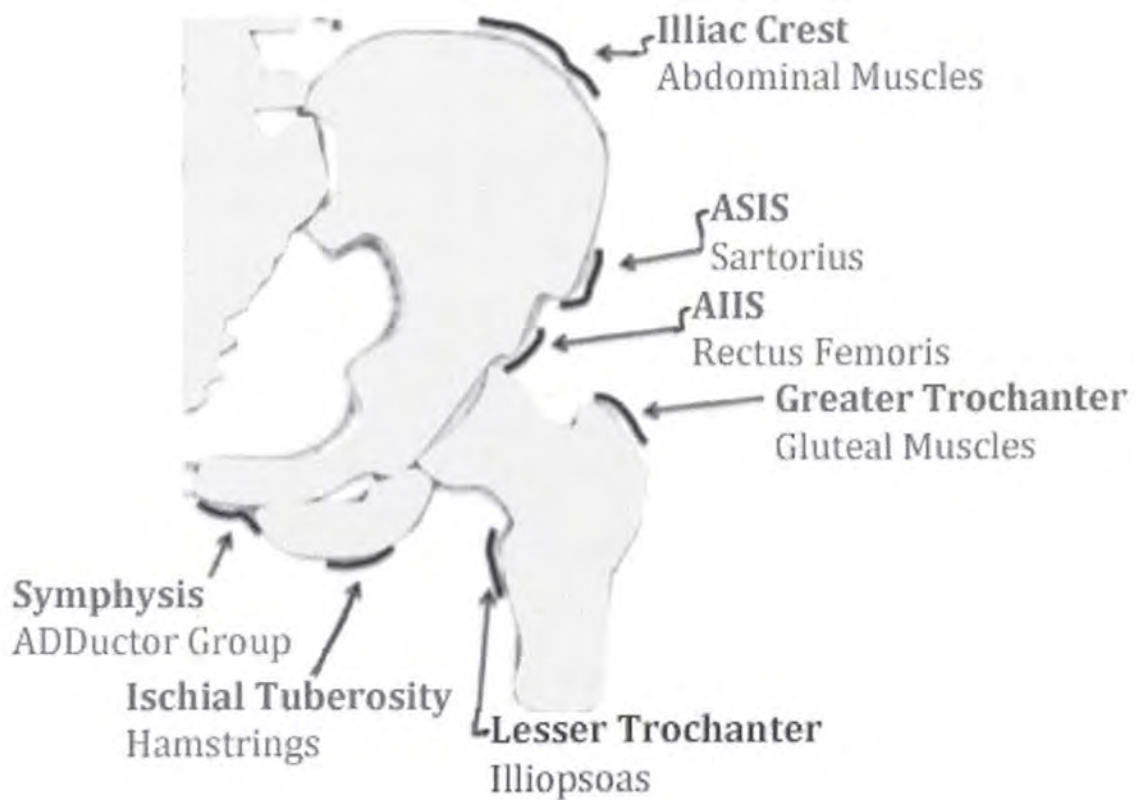
- o Iliopectineal Line = Anterior
- o Ilioischial Line = Posterior (remember you sit on your ischium)
- o The both column fracture by definition divides the ilium proximal to the hip joint, so you have no articular surface of the hip attached to the axial skeleton (that's a problem).

Corona Mortis: The anastomosis of the inferior epigastric and obturator vessels sometimes rides on the superior pubic ramus. During a lateral dissection - sometimes used to repair a hip fracture this can be injured. I talk about this more in the vascular chapter.

Hip Fracture Leading to AVN: The location of the fracture may predispose to AVN. It's important to remember that since the femoral head gets vascular flow from the circumflex femorals a **displaced intracapsular fracture could disrupt this blood supply - leading to AVN.**

- **Testable Point:** *Degree of fracture displacement corresponds with risk of AVN.*

Avulsion Injury: This is seen more in kids than adults. Adult bones are stronger than their tendons. In kids it's the other way around. One pearl is that if you see an **isolated “avulsion” of the lesser trochanter in a seemingly mild trauma / injury in an adult - query a pathologic fracture.** Now, to discuss what I believe is the highest yield topic in MSK for the CORE, “where did the avulsion come from?” The easiest way to show this is a plain film pelvis (or MRI) with a tug/avulsion injury to one of the muscular attachment sites. The question will most likely be “*what attaches there?*” or “*which muscle got avulsed?*”



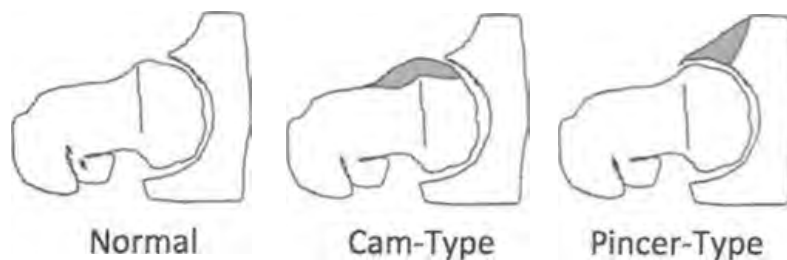
Snapping Hip Syndrome: The clinical sensation of “snapping” or “clicking” with hip flexion and extension. For a multiple choice testing standpoint all you need to know is (1) that it exists, (2) there are three types, and (3) what muscle each type involves.

Snapping Hip Syndrome	
External (most common)	Iliotibial Band over Greater Trochanter
Internal	Iliopsoas over Iliopectineal eminence or femoral head
Intra-Articular	Labral tears / joint bodies

Femoroacetabular Impingement (FAI): This is a syndrome of painful hip movement. It’s based on hip / femoral deformities, and honestly might be total BS. Supposedly it can lead to early degenerative changes. There are two described subtypes:

Pincer Impingement	Cam Impingement
Middle Aged Women	Young Man
Over Coverage of the femoral head by the acetabulum	Bony protrusion on the antero-superior femoral head-neck junction
“Cross Over Sign ”	“Pistol Grip Deformity ” Describes the appearance of the femur

Femoroacetabular Impingement



Memory Aid

I remember that the femoral one (cam-type) is more common in men because the femoral head kinda looks like a penis.

Be honest, you were thinking that too.

“Cross Over Sign ” - A sign of pincer type FAI. This refers to the anterior and posterior rims of the acetabulum forming a “figure of eight” on AP pelvis. It is extremely unreliable, and heavily based on positioning.

Sacrum:

You can get fractures of the sacrum in the setting of trauma, but if you get shown or asked anything about the sacrum it's going to be either (a) SI degenerative change - discussed later, (b) unilateral SI infection, (c) a chordoma - discussed later, (d) sacral agenesis, or (e) an insufficiency fracture. Out of these 5 things the insufficiency fracture is probably the most likely.

Sacral Insufficiency Fracture - The most common cause is postmenopausal **osteoporosis**. You can also see this in patients with renal failure, patients with RA, **pelvic radiation**, **mechanical changes after hip arthroplasty**, or extended steroid use. They are often (usually) occult on plain films. They will have to show this either with a bone scan, or MRI.



The classic “**Honda Sign**” from the “H” shaped appearance is probably the most likely presentation on a multiple choice test.

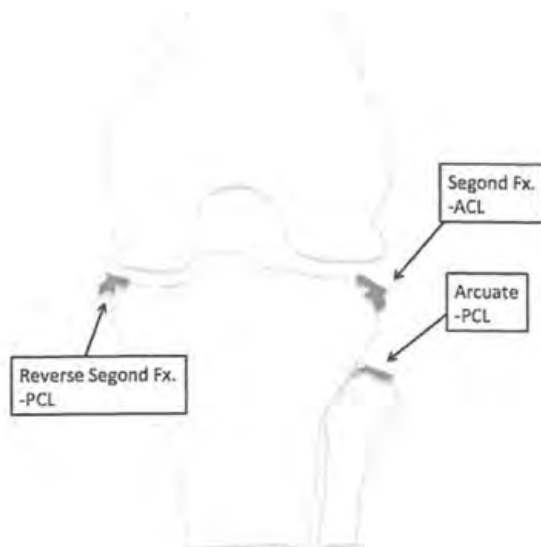
"Honda Sign"
Sacral Insufficiency Fx

Knee / Tibia / Fibula:

Second Fracture: This is a fracture of the **Lateral Tibial Plateau** (*common distractor is medial tibia*). The thing to know is that it is **associated with ACL tear (75%)**, and occurs with **internal rotation**.

Reverse Second Fracture: This is a fracture of the **Medial Tibial Plateau**. The thing to know is that it is **associated with a PCL tear**, and occurs with **external rotation**. There is also an associated **medial meniscus injury**.

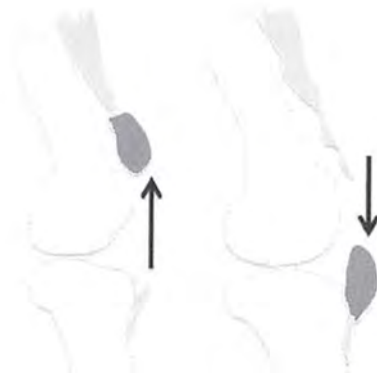
Arcuate Sign This is an avulsion of **proximal fibula** (insertion of arcuate ligament complex). The thing to know is that **90% are associated with cruciate ligament injury (usually PCL)**



Deep Intercondylar Notch Sign: This is a depression of the lateral femoral condyle (terminal sulcus) that occurs secondary to an impaction injury. This is **associated with ACL tears**.

Patella Dislocation: Basically **always lateral**, and the Medial Patello-Femoral Ligament is injured. There is a characteristic appearance on MRI, which will be shown and discussed later in this chapter.

Patella Alta / Baja: The patella will move up or down in certain traumatic situations. If the quadricep tendon tears you will get unopposed pull from the patellar tendon resulting in a low patella (Baja). If the patella tendon tears you will get unopposed quadriceps tendon pull resulting in a high patella (Alta).



Patella Alta Patella Baja

The “classic” association with patellar tendon tear (Alta) is **SLE**, (also can see in elderly, trauma, athletics, or RA). “Bilateral patellar rupture” is a buzzword for chronic steroids.

Tibial Plateau Fracture: This injury most commonly occurs from axial loading (falling and landing on a straight leg). The **lateral plateau is way more common than the medial**. If you see medial, it’s usually with lateral. Some dude named Schatzker managed to get the classification system named after him, of which type 2 is the most common (split and depressed lateral plateau).

Pilon Fracture (Tibial plafond fracture): This injury also most commonly occurs from axial loading, with the talus being driven into the tibial plafond. The fracture is characterized by comminution and articular impaction. About 75% of the time you are going to have fracture of the distal fibula.

Tibial Shaft Fracture: This is the most common long bone fracture. It was also *listed as the most highly tested subject in orthopedic OITE exam (with regard to trauma)*, over the last 8 years. Apparently there are a bunch of ways to put a nail or plate in it. It doesn’t seem like it could be that high yield for the CORE compared to other fractures with French or Latin sounding names.

Tillaux Fractures: This is a **salter-harris 3**, through the anterolateral aspect of the distal tibial epiphysis.

Triplane Fracture: This is a **salter-harris 4**, with a vertical component through the epiphysis, horizontal component through the physis, and oblique through the metaphysis.

Maisonneuve Fracture: This is an unstable fracture involving the medial tibial malleolus and/or **disruption of the distal tibiofibular syndesmosis**. The most common way to show this is to first show you the ankle with the widened mortis, and “*next step?*” get you to ask for the proximal fibula - which will show the **fracture of the proximal fibular shaft**. This fracture pattern is unique as the forces begin distally in the tibiotalar joint and ride up the syndesmosis to the proximal fibula. For some reason knowing that the fracture **does not extend into the hindfoot** is a piece of valuable trivia.



Maisonneuve Fracture:

Foot / Ankle

Casanova Fracture - If you see bilateral calcaneal fractures, you should “*next step?*” look at the spine for a compression or burst fracture. These tend to occur in axial loading patterns (possibly from jumping out a window to avoid an angry husband).

Bohler’s Angle - The line drawn between the anterior and posterior borders of the calcaneus on a lateral view. An angle less than 20, is concerning for a fracture.

Jones Fracture: This is a fracture at the base of the fifth metatarsal, 1.5cm distal to the tuberosity. These are placed in a non-weight bearing cast.

Avulsion Fracture of the 5th Metatarsal: This is more common than a Jones fracture. The classic history is a dancer. It may be **secondary to tug from the lateral cord of the plantar aponeurosis or peroneus brevis** (this is controversial).

Stress Fracture of the 5th Metatarsal: This is considered a high risk fracture (hard to heal).



LisFranc Injury: This is the **most common dislocation of the foot**. The Lisfranc joint is the articulation of the tarsals and metatarsal heads. This joint would make a good place to amputate if you were a surgeon assisting in the Napoleonic invasion of Russia. The LisFranc ligament connects the medial cuneiform to the 2nd metatarsal base on the plantar aspect. If the ligament goes out you can see two patterns: (1) “Homolateral” - everyone moves lateral, (2) “Divergent” - the 1st MT goes medial, the 2nd-5th MT goes lateral.

What you need to know:

- Can't exclude it on a non-weight bearing film
- Associated fractures are most common at the base of the 2nd MT - “*Fleck Sign*”
- Fracture non-union and post traumatic arthritis are gonna occur if you miss it (plus a lawsuit).

“*FleckSign*” - This is a small bony fragment in the LisFranc Space (between 1st MT and 2nd MT) - that is associated with an avulsion of the LF ligament.

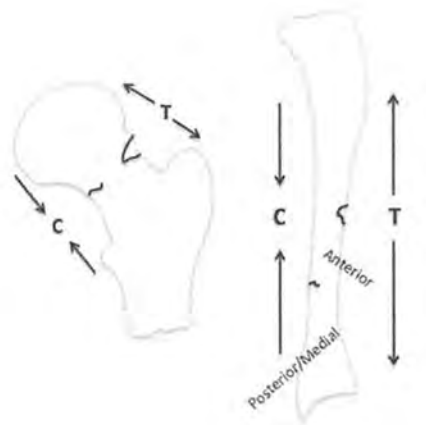
Spine: *Fractures and other acquired pathologies of the spine are discussed in detail in the spine section of the neuro chapter.*

Stress / Insufficiency Fractures:

Stress Fracture vs Insufficiency Fracture: A stress fracture is the result of abnormal stress on normal bone. An insufficiency fracture is the result of normal stress on abnormal bone.

Compressive Side vs Tensile Side: This comes up in two main areas - the femoral neck and the tibia. Fractures of the compressive side are constantly getting pushed back together - these do well. Fractures of the tensile side are constantly getting pulled apart - these are a pain in the ass to heal.

- **Femoral Stress Fracture:** Fractures along the compressive (medial) side are more common, typically seen in a younger person along the inferior femoral neck. Fractures along the tensile (lateral) side are more common in old people.



Compressive vs Tensile Forces
- Femoral Neck and Tibia

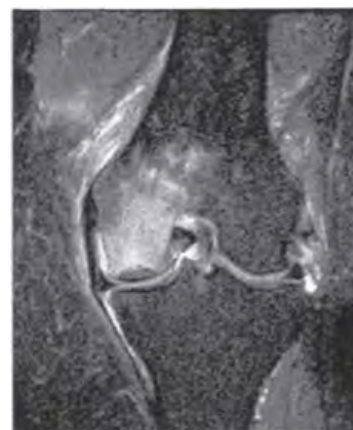
- **Tibial Stress Fracture:** This is the *most common site of a stress fracture in young athletes*. These are most common on the compressive side (posterior medial) in either the proximal or distal third. Less common are the tensile side (anterior) fractures, and these favor the mid shaft. They are bad news and don't heal - often called "*dreaded black lines*."

SONK (Spontaneous Osteonecrosis of the Knee):

This is totally named wrong, as it is another type of insufficiency fracture. You see this in old ladies with the classic history of "sudden pain after rising from a seated position." Young people can get it too (much less common), usually seen after a meniscal surgery.

Things to know about SONK:

- *It's an insufficiency fracture (NOT osteonecrosis)*
- *Favors the medial femoral condyle (area of maximum weight bearing)*
- *Usually unilateral in an old lady without history of trauma*
- *Associated with meniscal injury*



SONK
- Diffuse Increased Signal (Edema)

Navicular Stress Fracture - You see these in runners who run on hard surfaces. The thing to know is that just like in the wrist, the navicular is high risk for AVN.

March Fracture: This is a metatarsal stress fracture, which is fairly common. Classically seen in military recruits that are marching all day long.

Calcaneal Stress Fracture - The calcaneus is actually the most fractured tarsal bone. The fractures are usually intra-articular (75%). The stress fracture will be seen, with the fracture line perpendicular to the trabecular lines.

High Risk vs Low Risk Stress Fractures: You can sort these based on the likelihood of uncomplicated healing when treated conservatively.	
High Risk	Low Risk
Femoral Neck (tensile side)	Femoral Neck (compressive side)
Transverse Patellar Fracture	Longitudinal Patellar Fracture
Anterior Tibial Fracture (midshaft)	Posterior Medial Tibial Fracture
5 th Metatarsal	2 nd and 3 rd Metatarsal
Talus	Calcaneus
Tarsal Navicular	
Sesamoid Great Toe	

Osteoporosis / Osteopenia & Complications:

Osteopenia: This just means increased lucency of bones. Although this is most commonly caused by osteoporosis that is not always the case.

Osteomalacia: This is a soft bone from excessive uncalcified osteoid. This is typically related to vitamin D issues (either renal causes, liver causes, or other misc causes). It generally looks just like diffuse osteopenia. For the purpose of multiple choice you should think about 4 things; Ill-defined trabeculae, Ill-defined corticomedullary junction, bowing, and “Looser’s Zones.”

Looser Zones: These things are wide lucent bands that transverse bone at right angles to the cortex. You should think two things: **osteomalacia** and **rickets**. Less common is 01. The other piece of trivia is to understand **they are a type of insufficiency fracture**.

Osteoporosis: The idea is that you have low bone density. Bone density peaks around 30 and then decreases. It decreases faster in women during menopause. The imaging findings are a thin sharp cortex, prominent trabecular bars, lucent metaphyseal bands, and spotty lucencies.

Causes: Age is the big one. Medications (steroids, heparin, dilantin), Endocrine issues (cushings, hyperthyroidism), Anorexia, and Osteogenesis Imperfecta.

Complications: Fractures - Most commonly of the spine (2nd most common is the hip, 3rd most common is the wrist).

DEXA: This is a bone mineral density test and an excellent source of multiple choice trivia.

General Things to know about DEXA

- T score = Density relative to young adult
- T score defines osteopenia vs osteoporosis
- T score >1.0 = Normal, -1.0 to -2.5 = Osteopenia, <-2.5 **Osteoporosis**
- Z score = Density relative to aged match control “to Za Zame Age”
- False negative / positives (see below)

False Positive /Negative on DEXA: DEXA works by measuring the density. Anything that makes that higher or lower than normal can fool the machine.

False Positive:

- * Absent Normal Structures: Status post laminectomy

False Negative:

- * Including excessive Osteophytes, dermal calcifications, or metal
- * Including too much of the femoral shaft when doing a hip - can elevate the number as the shaft normally has denser bone.

Reflex Sympathetic Dystrophy (RSD):

Can cause severe osteopenia (like disuse osteopenia). Some people say it **looks like unilateral RA, with preserved joint spaces**. Hand and shoulder are most common sites of involvement. May occur after trauma or infection resulting in an overactive sympathetic system. It's one of the many causes of a 3 phase hot bone scan. In fact, *intra-articular uptake* of tracer on bone scan in patients with RSD (secondary to the increased vascularity of the synovial membrane), and this is somewhat characteristic.

Transient Osteoporosis:

There are two types of presentations.:

Transient osteoporosis of the hip: For the purpose of multiple choice tests by far you should expect to see the **female in the 3rd trimester of pregnancy** with involvement of the left hip. Having said that, it's actually more common in men in whom it's usually bilateral. The joint space should remain normal. It's self limiting (hence the word transient) and resolves in a few months. *Plain film shows osteopenia, MRI shows Edema, Bone scan shows increased uptake focally.*

Regional migratory osteoporosis - This is an idiopathic disorder which has a very classic history of **pain** in a joint, which gets better then shows up in another joint. It's associated with osteoporosis - which is also self limiting. It's more common in men.

Osteoporotic Compression Fracture: A band-like, horizontally elongated focus low T2 and low T1 -weighted signal intensity immediately underneath the fractured endplate is a feature of benign osteoporotic compression fractures. A similar band with T2 bright, and T1 dark can also be seen with osteoporotic fractures. The non-deformed portions of the vertebral body should have normal signal.

Neoplastic Compression Fracture: Most vertebral mets don't result in compression fracture until nearly the entire vertebral body is replaced with tumor. If you see abnormal marrow signal (not band like) with involvement of the posterior margin you should think about cancer. *Next step ?* - look at the rest of the spine - mets are often multiple.

OCDs / OCLs

Osteochondritis Dissecans (OCD): The new terminology is actually to call these "OCLs" (the "L" is for Lesion). This a spectrum of aseptic separation of an osteochondral fragment which can lead to gradual fragmentation of the articular surface and secondary OA. Most of the time it is secondary to trauma, although it could also be secondary to AVN.

Where it happens: Classic locations include the femoral condyle (most common site in the knee), patella, talus, and capitellum.

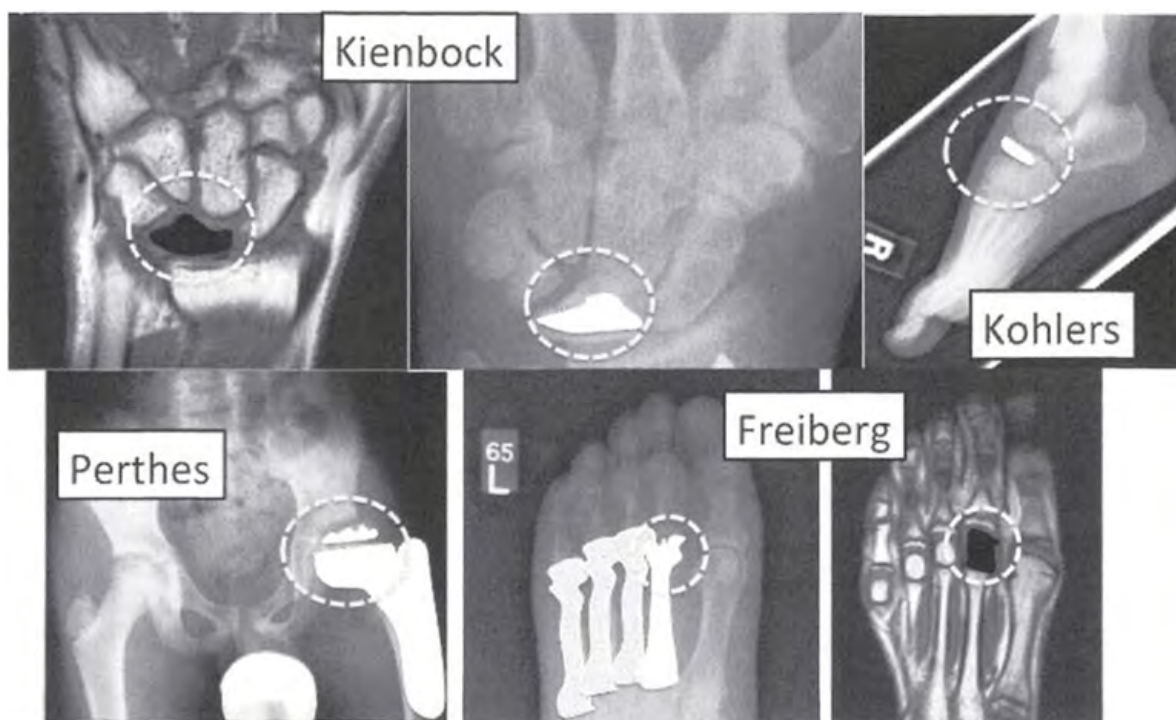
Staging: There is a staging system, which you probably need to know exists.

- # Stage 1: Stable - Covered by intact cartilage, Intact with Host Bone
- * Stage 2: Stable on Probing, Partially not intact with host bone.
- Stage 3: Unstable on Probing, Complete discontinuity of lesion.
- * Stage 4: Dislocated fragment

Treatment / Who cares? If the fragment is unstable you can get secondary OA. You want to **look for high T2 signal undercutting the fragment from the bone to call it unstable** (edema can force a false positive). Thus, the absence of high T2 signal at the bone fragment interface is a good indicator of osseous bridging and stability. Granulation tissue at the interface (which will enhance with Gd), does not mean it's stable.

Osteochondroses: These are a group of conditions (usually seen in childhood) that are characterized by involvement of the epiphysis, or apophysis with findings of collapse, sclerosis, and fragmentation - suggesting osteonecrosis.

Kohlers	Tarsal Navicular	Male 4-6. Treatment is not surgical.
Freiberg Infraction	Second Metatarsal Head	Adolescent Girls - can lead to secondary OA
Severs	Calcaneal Apophysis	Some say this is a normal "growing pain"
Panners	Capitellum	Kid 5-10 "Thrower" ; does not have loose bodies.
Perthes	Femoral Head	White kid; 4-8.
Kienbock	Carpal Lunate	Associated with negative ulnar variance. Seen in person 20-40.

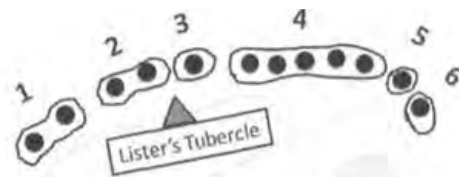


Soft Tissue Injury /Acquired (stuff likely shown with MRI)

MRI of the Wrist / Hand:

Don't panic if you are shown a MRI of the wrist, there are only a few things that they can show you. First let's briefly review the anatomy. With regard to the extensor tendons there are four things to know:

- There are 6 extensor compartments (5 fingers + 1 for good luck).
- First compartment (APL and EPB) are the ones affected in de Quervain's
- Third compartment has the EPL which courses beside Lister's Tubercle.
- The sixth compartment (Extensor Carpi Ulnaris) - can get an early tenosynovitis in rheumatoid arthritis.

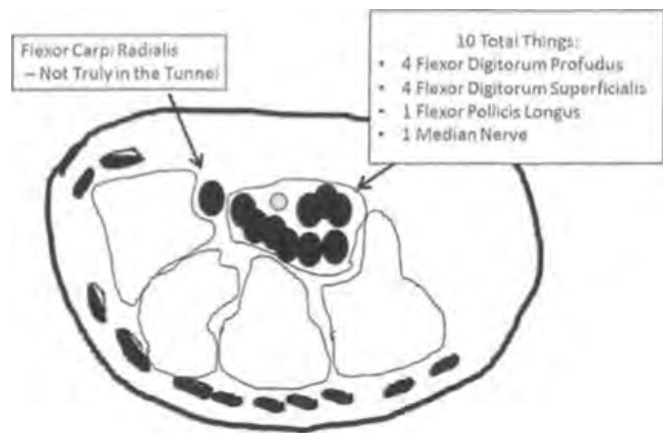


Extensor Compartments

Carpal Tunnel: They could show you the carpal tunnel, but only to ask you about anatomy.

*What goes through the carpal tunnel
(more easily asked as what does
NOT go through)?*

Knowing what is in (and not in) the carpal tunnel is high yield for multiple choice testing. The tunnel lies deep to the palmaris longus, and is defined by 4 bony prominences (pisiform, scaphoid tubercle, hook of hamate, trapezium tubercle), with the transverse carpal ligament wrapping the contents in a fibrous sheath



The tunnel contains 10 things (4 Flexor Profundus, 4 Flexor Superficials, 1 Flexor Pollicis Longus, and 1 Median Nerve). The Flexor Carpi Radialis is not truly in the tunnel. The extensor tendons are on the other side of the hand. Note that flexor pollicis longus goes through the tunnel, but flexor pollicis brevis does not (it's an intrinsic hand muscle).

Anatomic Trivia Regarding the Spaces of the Wrist:

Which synovial spaces normally communicate ? The answer is **pisiform recess and radiocarpal joint**. I can think of two ways to ask this (1) related to fluid - the bottom line is that excessive fluid in the pisiform recess should not be considered abnormal if there is a radiocarpal effusion, and (2) that either space can be used for wrist arthrography.

Other joint spaces in the body, easily lending to multiple choice testing:

Glenohumeral Joint and Subacromial Bursa	Should NOT communicate. Implies the presence of a full thickness rotator cuff tear.
Ankle Joint and Common (lateral) Peroneal Tendon Sheath	Should NOT communicate. Implies a tear of the calcaneofibular Ligament.
Achilles Tendon and Posterior Subtalar Joint	Should NOT communicate. The Achilles tendon does NOT have a true tendon sheath.
Pisiform Recess and Radiocarpal Joint	Should normally communicate.

Common Pathology Seen on MRI of the Wrist / Hand:

Triangular Fibrocartilage Tears: These can be acute or chronic. The acute one is going to be a young person with a tear on the ulnar side. The chronic one is more likely to be shown with ulnar abutment syndrome (positive ulnar variance with cystic change in the lunate). Degeneration of this cartilage is common (50% at age 60).

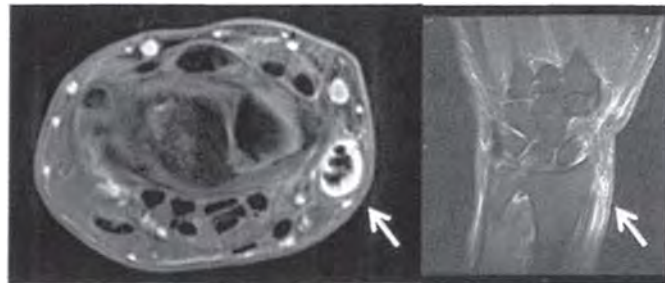
Scapholunate Ligament Tear: The Terry Thomas look on plain film. The low signal ligament will be disrupted. The bones will still be wide.

Kienbocks: AVN of the lunate, seen in people in their 20s-40s. *The most likely testable trivia is the association with negative ulnar variance.* It's going to show signal drop out on T1.

De Quervain's Tenosynovitis: This is the so called "Washer Woman's Sprain" or "Mommy Thumb" from repetitive activity / overuse. The classic history is "new mom - holding a baby." The affected area is the **first dorsal (extensor) compartment** (extensor pollicis brevis and abductor pollicis longus). This is way more common in women. The presence or absence of an intratendinous septum is a prognostic factor.

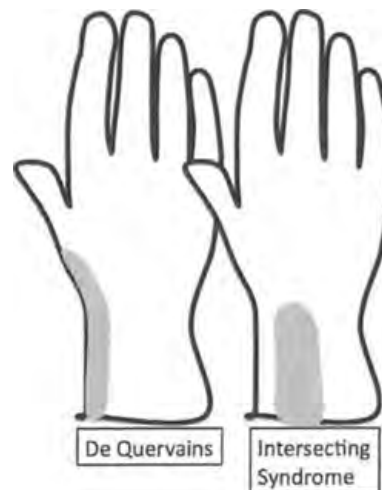
How it s shown:

- * *This can be shown with ultrasound, as increased fluid within the first extensor tendon compartment*
- * *This can be shown with MRI, as increased T2 signal in the tendon sheath*



DeQuervain's Syndrome

Intersection Syndrome: A repetitive use issue (classically *seen in rowers*), where the first extensor compartment, cross over those of the second extensor compartment. The result is extensor carpi radialis brevis and longus tenosynovitis .



Tenosynovitis : This is an inflammation of the tendon, with increased fluid seen around the tendon. This will be shown on MRI.

Diffuse:

- **Nontuberculous Mycobacterial Infection** - The hand and the wrist are the most common areas affected. This is a diffuse exuberant tenosynovitis that spares the muscles. It usually occurs in patients who are immunocompromised.

Rheumatoid Arthritis: A nice trick is to show that multiple flexor tendons , or extensor carpi ulnaris - tenosynovitis can present as an early RA (before bone findings).

Focal:

Overuse: This is going to be classic locations like 1st extensor compartment for De Quervains.

- **Infection:** The hard rule is that tenosynovitis of any flexor tendon is a surgical emergency as it can spread rapidly to the common flexors of the wrist. You can get increased pressures and necrosis of the tendons. Delayed treatment tend to do terrible.

Gamesmanship Wrist Compartments	
Isolated 1st	De Quervains
1 st + 2nd	Intersecting
Isolated 6th	Early RA
Multiple Flexors	RA

Finger Tumor: They can show scalloping on a plain film of the finger, but if they want a single diagnosis they need to show a MRI. There is a long differential, but the only things they are going to show are:

- **Glomus Tumor:** This is a benign vascular tumor seen at the tips of fingers (75% in hand). It will be T1 low, **T2 bright**, and **enhance avidly**.
 - **Giant Cell Tumor of the Tendon Sheath:** This is **basically PVNS of the tendon**. Typically found in the hand (palmar tendons). Can cause erosions on the underlying bone. Will be soft tissue density, and be **T1 and T2 dark** (contrasted to a glomus tumor which is T1 dark, T2 bright, and will enhance uniformly). **Will bloom on gradient**.
- * **Fibroma:** This is a benign overgrowth of the tendon collagen. It's going to be low on T1 and low on T2. **Will NOT bloom like a GCT will**.

Finger Tip Tumors / Masses		
Glomus	T1 Dark, T2 Bright, Enhances avidly.	T2 Bright, Enhance Avidly.
Giant Cell Tumor Tendon	T1 Dark, T2 Dark, Variable Enhancement, Bloom on Gradient	Bloom on Gradient
Fibroma	T1 Dark, T2 Dark. No Blooming	Does NOT Bloom on Gradient.

Dupuytren Contracture: This is the most common of the fibromatoses. The classic scenario is a white person from North Europe with alcoholic liver disease. It's a nodular mass on the palmar aspect of the aponeurosis that progresses to cord-like thickening and eventual contracture (usually involving the 4th finger). It's bilateral about half the time.

Common Pathology Seen on MRI of the Elbow:

If you are shown an MRI of the Elbow don't panic, there are only a few things they can show you.

Cubital Tunnel Syndrome

- The result of repetitive valgus stress
- *Anatomy Trivia:* the site where the ulnar nerve passes beneath the cubital tunnel retinaculum also known as the epicondylo-olecranon ligament or Osborne band
- Can occur from compression by any pathology (tumor, hematoma, etc...), when it occurs from an **accessory muscle** it's classically the **anconeus epitrochlearis**

Partial Ulnar Collateral Ligament Tear:

For the CORE exam all you really need to know is that throwers hurt their ulnar collateral ligament (which attaches on the medial coronoid - *sublime tubercle*). The ligament has three bundles, and the **anterior bundle is by far the most important**. If you get any images it is most likely going to be of the partial UCL tear, described as the “**T sign**,” with contrast material extending medial to the tubercle.



Normal T-Sign

"UCL Partial Tear"

Panner Disease: This is one of the osteochondroses of the capitellum. It's seen in kids 5-10, and thought to be related to trauma from throwing (baseball playing). It looks a lot like an OCD lesion of the elbow (which also favors the capitellum).

Panner	Osteochondritis Dissecans
Affects the Capitellum	Also favors the Capitellum
Age 5-10	Teenager
Low T1, High T2	Low T1, High T2
No Loose Bodies	Loose Bodies

Lateral Epicondylitis (*more common than medial*) - *seen in Tennis Players* -

- Extensor Tendon Injury (classically extensor carpi radialis brevis)
- Radial Collateral Ligament Complex - Tears due to varus stress

Medial Epicondylitis (*less common than lateral*) - *seen in golfers*

- Common flexor tendon and ulnar nerve may enlarge from chronic injury

Epitrochlear Lymphadenopathy - This is a classic look for cat-scratch disease.

Dialysis Elbow: This is the result of olecranon bursitis from constant pressure on the area, related to positioning of the arm during treatment.

Common Pathology Seen on MRI of the Shoulder:

Impingement / Rotator Cuff Tears: This is a high yield / confusing subject that is worth talking about in a little more detail. In general, rotator cuff pathology is the result of overuse activity (sports) or impingement mechanisms. There are two types of impingement with two major sub-divisions within those types. Like many things in Radiology if you get the vocabulary down, the pathology is easy to understand.

External: This refers to impingement of the rotator cuff overlying the bursal surfaces (superficial surfaces) that are adjacent to the coracoacromial arch. As a reminder the arch is made up of the coracoid process, acromion, and coracoacromial ligament.

Primary External Causes (Abnormal Coracoacromial Arch) :

- The **hooked acromion** (type III Bigliani) is more associated with external impingement than the curved or flat types.
- **Subacromial osteophyte formation** or thickening of the coracoacromial ligament
- **Subcoracoid impingement:** Impingement of the subscapularis between the coracoid process and lesser tuberosity. This can be secondary to congenital configuration, or a configuration developed post traumatically after fracture of the coracoid or lesser tuberosity.

Secondary External Causes (Normal Coracoacromial Arch):

- **“Multidirectional Glenohumeral Instability”** - resulting in micro-subluxation of the humeral head in the glenoid, resulting in repeated microtrauma. The important thing to know is this is *typically seen in patients with generalized joint laxity*, often involving both shoulders.

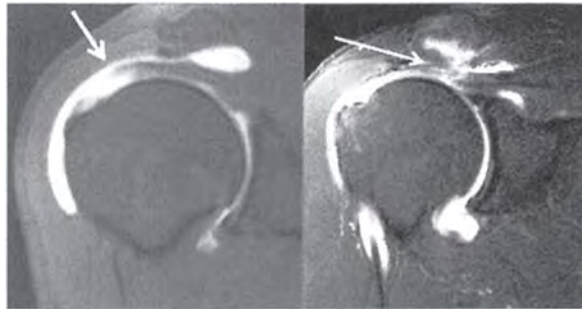
Internal: This refers to impingement of the rotator cuff on the undersurface (deep surface) along the glenoid labrum and humeral head.

- **Posterior Superior:** This is a type of impingement that occurs when the posterior superior rotator cuff (junction of the supra and infraspinatus tendons) comes into contact with the posterior superior glenoid. Best seen in the ABER position, where these tendons get pinched between the labrum and greater tuberosity. This is seen in athletes who make overhead movements (throwers, tennis, swimming).
- **Anterior Superior:** This is internal impingement that occurs when the arm is in horizontal adduction and internal rotation. In this position, the undersurface of the biceps and subscapularis tendon may impinge against the anterior superior glenoid rim.

High Yield Trivia Points on Impingement	
Subacromial Impingement - most common form, resulting from attrition of the coracoacromial arch.	Damages Supraspinatus Tendon .
Subcoracoid Impingement - Lesser tuberosity and coracoid do the pinching.	Damages Subscapularis
Posterior Superior Internal Impingement - Athletes who make overhead movements. Greater tuberosity and posterior inferior labrum do the pinching.	Damage Infraspinatus (and posterior fibers of the supraspinatus).

Rotator Cuff Tears: A tear of the articular surface is more common (3x more) than the bursal surface. The underlying mechanism is usually degenerative, although trauma can certainly play a role. The terminology “massive rotator cuff tear” refers to at least 2 out of the 4 rotator cuff muscles. The **most common of the four muscles to tear is the Supraspinatus**. The teres minor is the least common to tear. **A partial tear that is > 50% is what the surgeon wants to know.** A final general piece of trivia is that a tear of the fibrous rotator cuff interval (junction between anterior fibers of the Supraspinatus and superior fibers of the subscapularis), is still considered a rotator cuff tear.

How do you know it's a full thickness tear?
You will have high T2 signal in the expected location of the tendon, with **Gad in the bursa.**



Full Thickness Rotator Cuff Tear
- Fluid / Gd Crossing over the cuff

Injury to the Labrum:

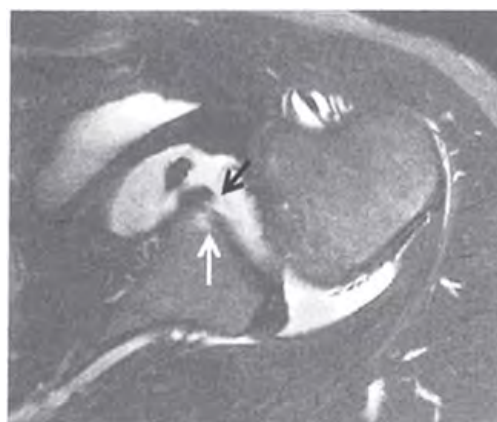
SLAP: Labral tears favor the superior margin and track anterior to posterior. As this tear affects the labrum at the insertion of the long head of the biceps, injury to this tendon is associated and part of the grading system (type 4).

Things to know about SLAP tears:

- Detachment of the superior labrum and biceps from the glenoid gets surgery (type 2)
- People under 40 usually have Bankart lesions
- People over 40 usually have Rotator Cuff Tears
- **NOT associated with Instability (usually)**

Mimics of SLAP Tears	
Sub Labral Sulcus	High Signal into the labrum is a tear. High signal along the normal contour is a sulcus
Sub Labral Foramen	High signal between the glenoid and labrum posterior to the attachment of the biceps is a tear

Bankart Lesions: There is an alphabet soup of Bankart related injuries, for the purpose of the CORE exam it seems that just understanding the spectrum (and what they are) is probably higher yield then the actual practical ability to differentiate these things.



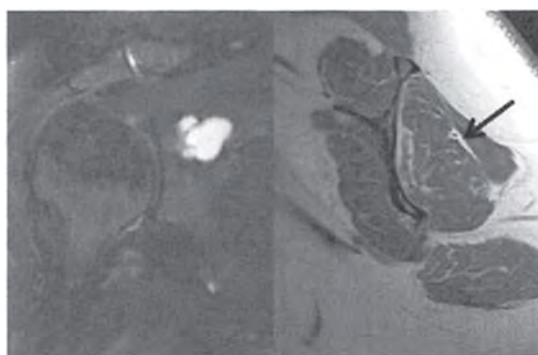
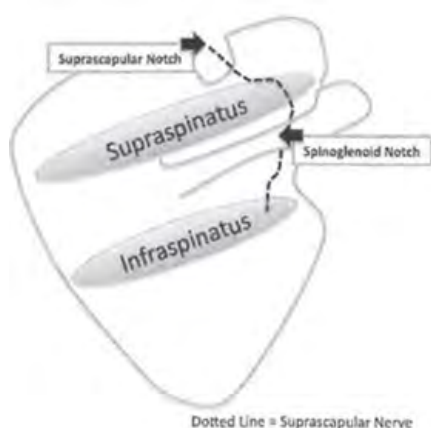
True Bankart
-The periosteum is disrupted

GLAD	Perthes	ALPSA	True Bankart
Superficial partial labral injury with cartilage defect	Avulsed anterior labrum (only minimally displaced). Inferior GH complex still attached to periosteum	Similar to perthes but with “bunched up” medially displaced inferior GH complex	Tom labrum
No instability	Intact Periosteum (lifted up)	Intact Periosteum	<i>Periosteum Disrupted</i>

HAGL: A non Bankart lesion that is frequently tested is the HAGL (Humeral avulsion glenohumeral ligament). This is an **avulsion of the inferior glenohumeral ligament**, and is most often the result of an anterior shoulder dislocation (just like all the above bankarts). The “J Sign” occurs when the normal U-shaped inferior glenohumeral recess is retracted away from the humerus appearing as a J.

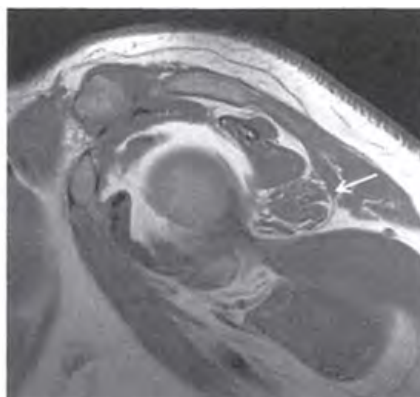
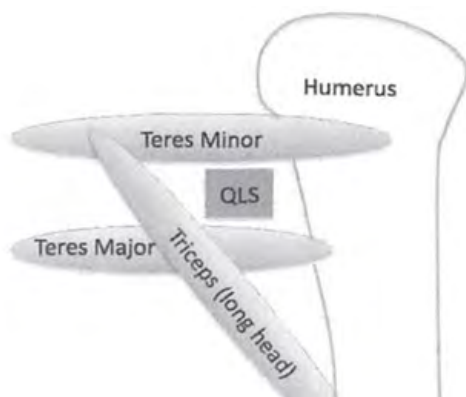
Nerve Entrapment: *High Yield Trivia:*

Suprascapular Notch vs Spinoglenoid Notch: A cyst at the level of the suprascapular notch will affect the supraspinatus and the infraspinatus. At the level of the spinoglenoid notch it will only affect the infraspinatus.



Cyst in the spinoglenoid notch causing fatty atrophy of the Infraspinatus

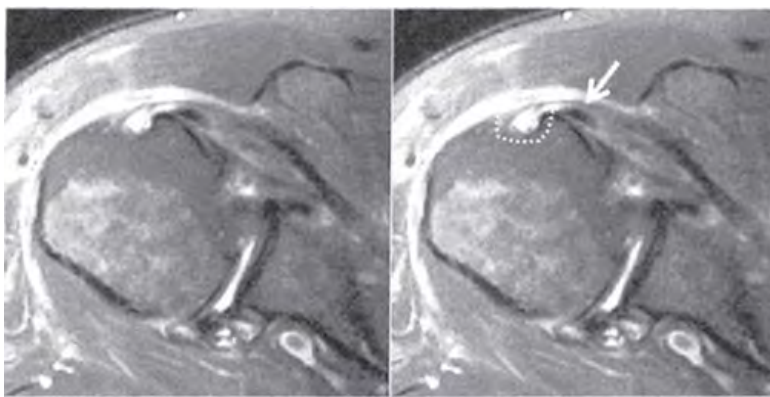
Quadrilateral Space Syndrome: Compression of the Axillary Nerve in the Quadrilateral Space (usually from fibrotic bands). They will likely show this with **atrophy of the teres minor**. Another classic question is to name the borders of the quadrilateral space: Teres Minor Above, Teres Major Below, Humeral neck lateral, and Triceps medial.



Quadrilateral Space Syndrome
-Atrophy of Teres Minor

Parsonage-Turner Syndrome: Think about this when you see muscles affected by pathology in two or more nerve distributions (suprascapular and axillary etc..). The condition is an idiopathic involvement of the brachial plexus.

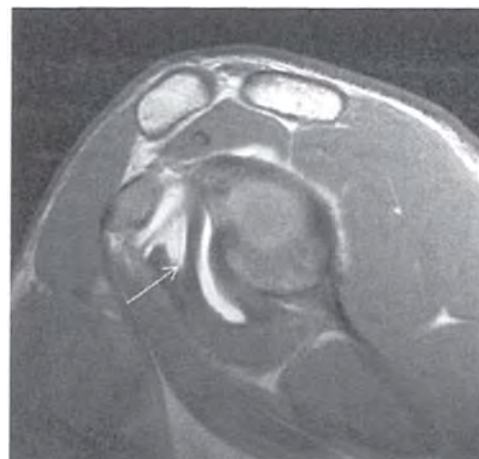
Subluxation of the Biceps Tendon: The subscapularis attaches to the lesser tuberosity. It sends a few fibers across the bicipital groove to the greater tuberosity, which is called the “transverse ligament”. A tear of the subscapularis opens these fibers up and allows the biceps to dislocate (usually medial). **Subscapularis Tear = Medial Dislocation of the Long Head of the Biceps Tendon.**



Subluxation of Biceps Tendon

Occurs with Tear of the Subscapularis

Buford Complex: A commonly tested (and occasionally seen) variant is the Buford Complex. This consists of an **absent anterior/superior labrum, along with a thickened middle glenohumeral ligament.**



Buford Complex
-Thick Middle GH Ligament

MRI of the Knee

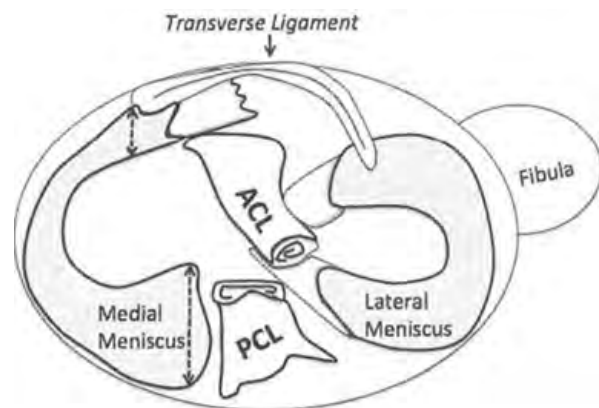
I want to focus on how the ABR is likely to ask the questions with two main pathways: (1) Total Trivia (2) Aunt Minnie Images

Anatomy:

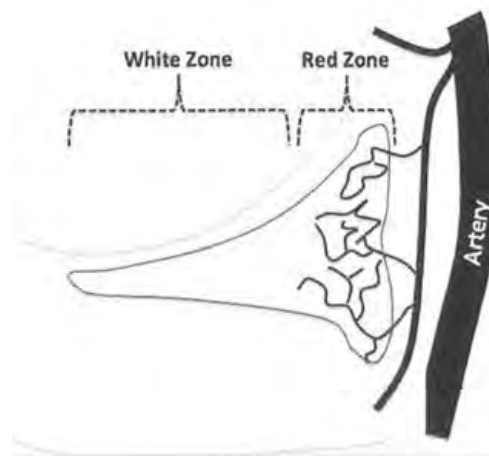
Ligaments: The ACL has two bundles. The long one (anteromedial) tightens the knee in flexion. The short one (posterior lateral) tightens the knee in extension. The PCL is the strongest ligament in the knee (you don't want a posterior dislocation of your knee resulting in dissection of your popliteal artery).

Meniscus: The meniscus is "C shaped", thick along the periphery and thin centrally. There are two main things to know about the meniscus:

- * Medial meniscus is thicker posterior. Lateral meniscus has equal thickness between anterior and posterior portion.



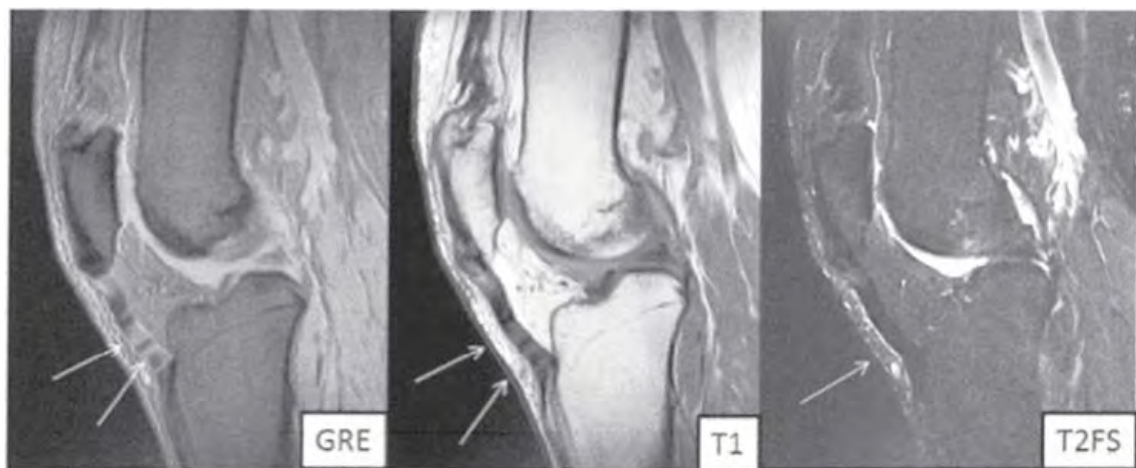
- The Peripheral "Red Zone" is vascular and might heal. The Central "white zone" is avascular and will not heal.



- * There are two menisiofemoral ligaments (Wrisberg, Humphry) which can be mimics of meniscal tears. Wrisberg is in the back ("*humping Humphry*"). You could also remember that "H" comes before "W" in the alphabet.

Tendons:

- The conjoint tendon is formed by the biceps femoris tendon and the LCL.
- The PCL and Patellar tendon may have foci of intermediate signal intensity on sagittal images with short echo time (TE) sequences where the tendon forms an angle of 55 degrees with the main magnetic field (*magic angle phenomenon*). This will NOT be seen on T2 sequences (with long TE). This phenomenon is reduced at higher field strengths due to greater shortening of T2 relaxation times.



Magic Angle: You see it on short TE sequences (T1, PD, GRE).
It goes away on T2.

Pathology:

Meniscal Tears: The peripheral meniscus (red zone) has better vasculature than the inner 2/3s (white zone) and might heal on its own. Broadly you can think about tears as either vertical or horizontal. Vertical tears can be sub divided into radial and longitudinal.



Vertical - Radial



Vertical - Longitudinal



Horizontal

Meniscal cysts are most often seen near the lateral meniscus and are often associated with horizontal cleavage tears.

Meniscocapsular Separation: The deepest layer of the **MCL** complex (capsular ligament) is relatively weak and is the first to tear; therefore there is an association with meniscocapsular separation.

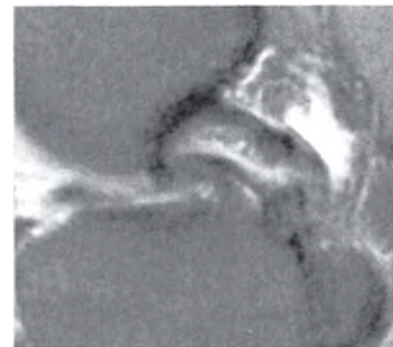
Discoid Meniscus: This is a normal variant of the **lateral meniscus** that is **prone to tear**. It's not C-shaped, but instead shaped like a disc. In other words, it's too big (too many bow-ties!).

There are three types, with the most rare and most prone to injury being the *Wrisberg Variant*.



Discoid Meniscus

Bucket Handle Tear: This is a torn meniscus (usually **medial** - 80%), that flips medially to lie anterior to the PCL. The classic Aunt Minnie appearance is that of a “**double PCL**.” Another piece of trivia is that a double PCL can **only occur in the setting of an intact ACL**, otherwise it won't flip that way. Just know it sorta indirectly proves the ACL is intact (I can just see some knucklehead asking that).



Double PCL

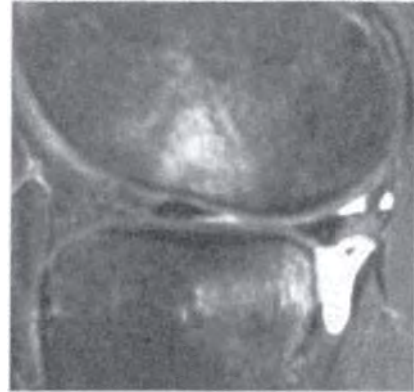
-Bucket Handle Meniscal Tear

Meniscal Ossicle: This is a focal ossification of the posterior horn of the lateral meniscus, that can be secondary to trauma or simply developmental.

ACL Tear: ACL tears happen all the time, usually in people who are stopping and pivoting.

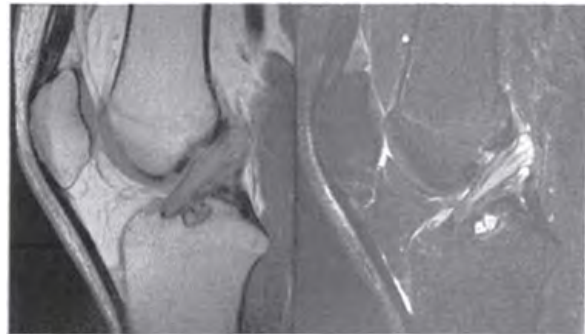
Things to know about ACL tears:

- * Associated with Segond Fracture
- * ACL Angle lesser than Blumensaat's Line
- * **O'donoghue's Unhappy Triad: ACL Tear, MCL Tear, Medial Meniscal**
- * *Classic Kissing Contusion Pattern:* The lateral femoral condyle (sulcus terminalis) bangs into the posterior lateral tibial plateau. This is 95% specific in adults.



ACL Tear
"Kissing Contusion Pattern"

ACL Muroid Degeneration: This can mimic acute or chronic partial tear of the ACL. There will be no secondary signs of injury (contusion etc..). It predisposes to ACL **ganglion cysts**, and they are usually seen together. The **T2/STIR buzzword is "celery stalk"** because of the striated look. The T1 **buzzword is "drumstick"** because it looks like a drum stick.



Muroid Degeneration of ACL
- "Drumstick / Celery Stick"

ACL Repair:

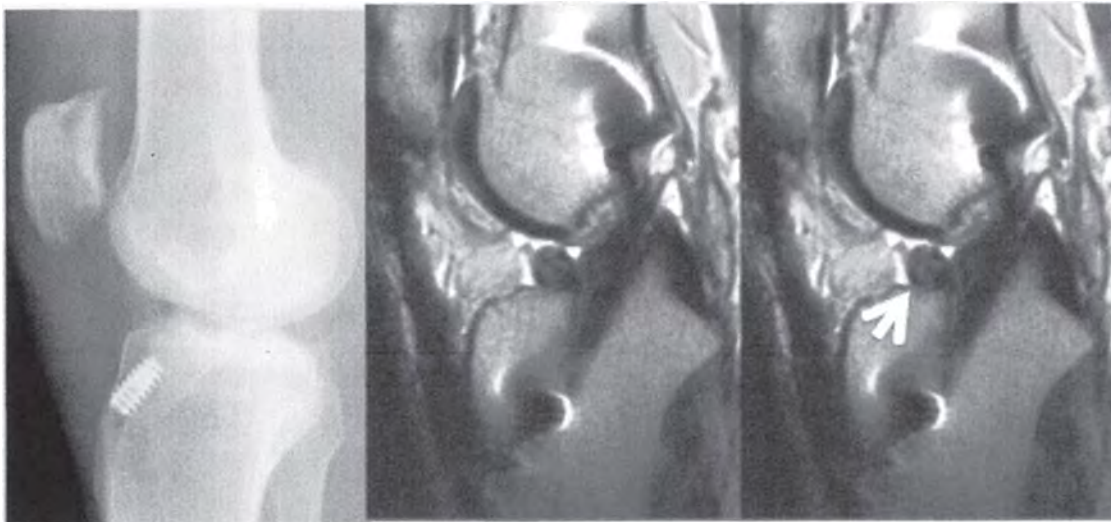
- Method 1: Using the middle one-third of the patellar tendon, with the patella bone plug attached to one end and tibial bone plug attached at the other.
- Method 2: Use a four-strand hamstring graft often made of semitendinosus or gracilis tendon, or both. Then fold and braid the segment to form a quadruple-thickness structure. The graft is then attached with interference screws, endobuttons, or staples. There is a lower reported morbidity related to harvest site using this method.

Complications:

“Roof Impingement” - If the tibial tunnel is placed too far anterior (partially or completely anterior to the intersection of the Blumensaat line), the graft may bump up against the anterior inferior margin of the intercondylar roof. The **positioning of the tibial tunnel is the primary factor in preventing impingement.**

“Maintaining Isometry” - “Isometry” is a word Orthopods use to define constant length and tension of the graft during full range of motion. Positioning of the **femoral tunnel** is the primary factor in maintaining isometry.

“Arthrofibrosis” Can be focal or diffuse (focal is more common). The focal form is the so called **“Cyclops” lesion** - so named because of its arthroscopic appearance. It’s gonna be a low signal speculated mass-like scar in Hoffa’s fat pad. It’s bad because it limits extension.



Cyclops Lesion - Scar associated with ventral graft

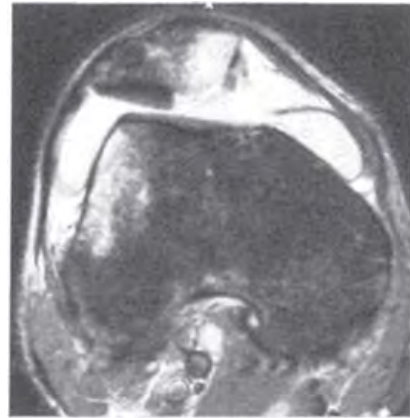
“Graft Tear” The graft is most susceptible to tear in the remodeling process (4-8 months post op). Signs of graft tear are increased T2 signal, and fiber discontinuity. Uncovering of the posterior horn of the lateral meniscus, and anterior tibial translation are considered good secondary signs.

PCL Tear: The posterior collateral ligament is the strongest ligament in the knee. A tear is actually uncommon, but you should think about it with a posterior dislocation.

Patella Dislocation - Dislocation of the patella is usually lateral because of the shape of the patella and femur. The contusion pattern is classic.

Things to know about Patella Dislocation:

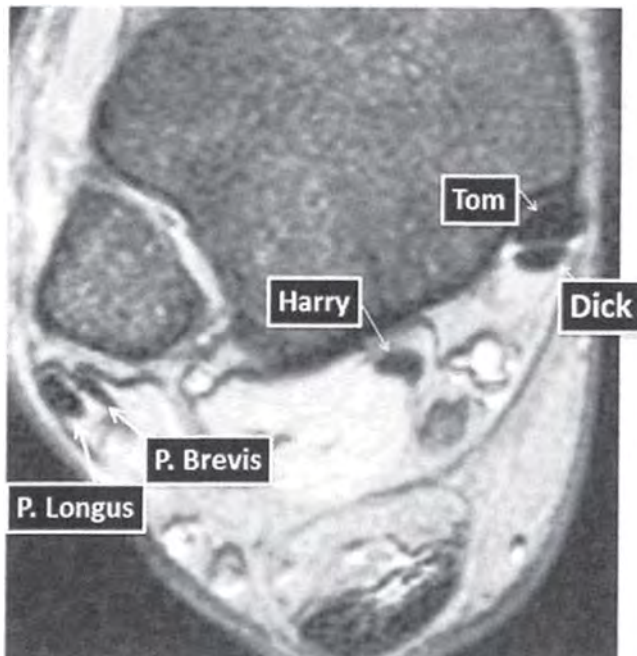
- It's Lateral
- Contusion Pattern - Classic
- Associated tear of the MPFL (medial patellar femoral ligament)



Patella Dislocation
Classic Contusion Pattern

The Foot / Ankle:

I'll lead with the most likely anatomic trivia, which is the tendons at the medial and lateral ankle.



Medial Ankle:

- Tom = Post Tibial
- Dick = Flex Digitorum
- Harry = Flex Hallucis

Lateral Ankle:

- Peroneus Longus
- Peroneus Brevis

The Mythical Master Knot of Henry - This has a funny sounding name, therefore it's high yield. This is where Dick (FDL) crosses Harry (FHL) at the medial ankle.

Whats the Master Knot of Henry? It's a "Harry Dick"

Common Pathology Seen on MRI of the Ankle / Foot:

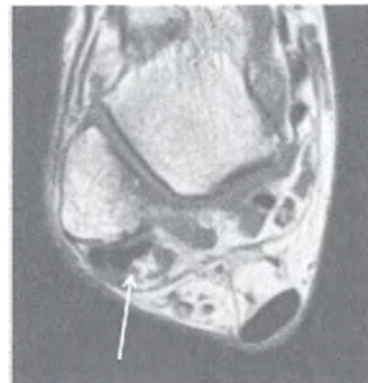
Ligamentous Injury: The highest yield fact is that the **anterior talofibular ligament is the weakest ligament and the most frequently injured** (usually from inversion).

Posterior Tibial Tendon Injury / Dysfunction: This results in a progressive flat foot deformity, as the PTT is the primary stabilizer of the longitudinal arch. When chronic, the tear is most common behind the medial malleolus (this is where the most friction is). When acute, the tear is most common at the insertion into the navicular bone. **Acute Flat Arch should make you think of PTT tear.** You will also have a hindfoot valgus deformity (from unopposed peroneal brevis action). The other point of trivia to know is that the spring ligament is a secondary supporter of the arch (it holds up the talar head), and it will thicken and degenerate without the help of the PTT. Don't get it twisted though, the spring ligament is very thick and strong and almost never ruptures in a foot/ankle trauma.

I Say Acute Flat Foot, You Say Posterior Tibial Tendon Injury

I Say PTT is out, You Say Spring Ligament is Thickened, and Hindfoot Valgus

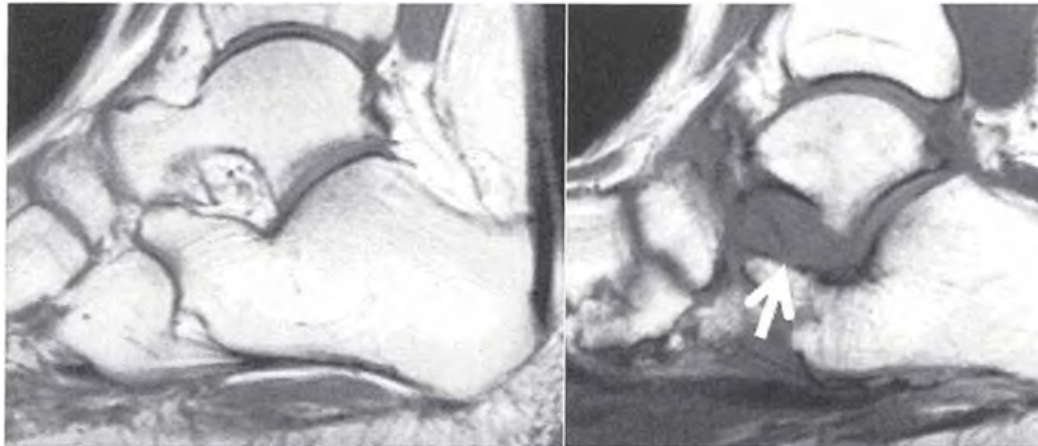
Split Peroneus Brevis: You can see longitudinal splits in the peroneus in people with inversion injuries. The history is usually "chronic ankle pain". The tendon will be C shaped or **boomerang shaped** with central thinning and partial envelopment of the peroneus longus. Alternatively, there may be 3 instead of 2 tendons. The tear occurs at the lateral malleolus. There is a strong (80%) association with lateral ligament injury.



Split Peroneus Brevis

Anterolateral Impingement Syndrome: Injury to the anterior talofibular ligaments and tibiofibular ligaments (usually from an inversion injury) can cause lateral instability, and chronic synovial inflammation. You can eventually produce a "mass" of hypertrophic synovial tissue in the lateral gutter. The **MRI finding is a "meniscoid mass" in the lateral gutter of the ankle**, which is a balled up scar (T1 and T2 dark).

Sinus Tarsi Syndrome: The space between the lateral talus and calcaneus. The syndrome is caused by hemorrhage or inflammation of the synovial recess with or without tears of the associated ligaments (talocalcaneal ligaments, inferior extensor retinaculum). There are associations with rheumatologic disorders and abnormal loading (flat foot in the setting of a posterior tibial tendon tear). The **MRI finding is obliteration of fat in the sinus tarsi space**, and replacement with scar.



Normal Sinus Tarsi
-Full of Fat

Sinus Tarsi Syndrome
-Full of Scar

Tarsal Tunnel Syndrome: Pain in the distribution of the tibial nerve (first 3 toes), from compression as it passes through the tarsal tunnel (behind the medial malleolus). It's usually unilateral (unlike carpal tunnel which is usually bilateral).

Achilles Tendon Injury: Acute rupture is usually obvious. The ability to plantar flex should be lost on exam (*unless plantaris muscle is intact - a common trick question*).

Xanthoma: Think about a xanthoma if the Achilles tendon is really enlarged / fusiform thickened. This can be seen in people with familial hypercholesterolemia, and is often bilateral.

Plantar Fasciitis: This is an inflammation of the fascia secondary to repetitive trauma. The pain is localized to the origin of the plantar fascia, and worsened by dorsiflexion of the toes. Buzzword is “*most severe in the morning.*” Plain film might show heel spurs, MRI may show a thickened fascia (> 4mm), with increased T2 signal, most significant near its insertion at the heel. A bone scan may show increased tracer in the region of the calcaneus (from periosteal inflammation).



Plantar Fasciitis

Morton's Neuroma: Soft tissue mass shown between the 3rd and 4th metatarsal heads is most likely a Morton's Neuroma (especially on multiple choice tests). They classically show it on short axis, T1 (it will be dark). The proposed pathology results from compression / entrapment of the plantar digital nerve in this location by the intermetatarsal ligament. Over time this results in thickening and development of perineural fibrosis.



Morton's Neuroma

- Dumbbell Scar Between 3rd and 4th Metatarsals

It's a big stupid scar, that looks like a dumbbell between the 3rd and 4th metatarsals. It's usually unilateral in a women.

Infection

Infection

With regard to osteomyelitis, radiographs will be normal for 7-10 days. Essentially, osteomyelitis can have any appearance, occur in any location, and at any age. Children have hematogenous spread usually hitting the long bones (metaphysis). Adults are more likely to have direct spread (in diabetic). However, you can have hematogenous spread in certain situations as well. General rule is that septic joints are more common in adults, osteomyelitis is more common in kids.

Knee Jerks:

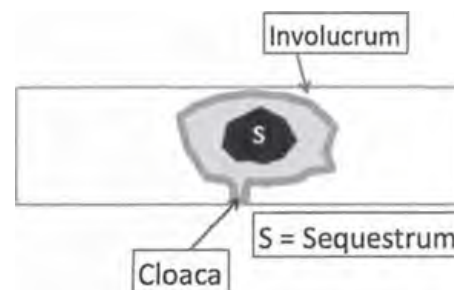
- * Osteomyelitis in Spine = IV Drug User
- * Osteomyelitis in Spine with Kyphosis = Gibbus Deformity = TD
- * Unilateral SI joint = IV Drug User
- * Psoas Muscle Abscess = TB

Hallmarks are destruction of bone and periosteal new bone formation.

Brodie's Abscess is a chronic infection (bone abscess). It's usually well circumscribed. It may have an osseous sequestrum (piece of necrotic bone surrounded by granulation tissue). As mentioned above, a sequestration has a DDx (Osteomyelitis, EG, Lymphoma, Fibrosarcoma).

Some frequently tested vocabulary:

- Sequestrum = Piece of necrotic bone surrounded by granulation tissue
- Involucrum = Thick sheath of periosteal bone around sequestrum
- Cloaca = The space / tract where the dead bone lives



Acute bacterial osteomyelitis can be thought of in three different categories: 1) hematogenous seeding (*most common in child*), 2) contiguous spread, and 3) direct inoculation of the bone either from surgery or trauma.

Acute hematogenous osteomyelitis has a predilection for the long bones of the body, specifically the metaphysis, which has the best blood flow and allows for spreading of the infection via small channels in the bone that lead to the subperiosteal space.

More Trivia that Multiple Choice Writers Love:

- * Age < 1 month = Multicentric involvement, often with joint involvement
 - o Bone scan often negative (75%) at this age
- * Age < 18 months = Spread to epiphysis through blood
- * Age 2-16 years = Trans-physeal vessels are closed (primary focus is metaphysis).

In the slightly older baby (<18 months) these vessels from the metaphysis to the epiphysis atrophy and the growth plate stops the spread (although spread can still occur). This creates a “septic tank” effect. This same thing happens with certain cancers (leukemia); the garbage gets stuck in the septic tank (metaphysis). Once the growth plates fuse, this obstruction is no longer present.

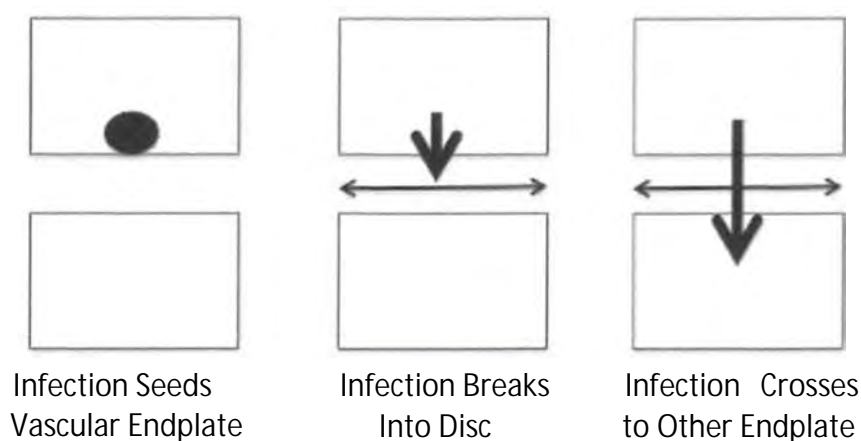
MRI findings of osteomyelitis: Low signal in the bone marrow on T1 imaging adjacent to an ulcer or cellulitis is diagnostic.

The Ghost Sign: Neuropathic Bone vs Osteomyelitis in a Neuropathic Bone

A bone that becomes a ghost (poor definition of margins) on T1 imaging, but then reappears (more morphologically distinct) on T2, or after giving IV contrast is more likely to have osteomyelitis.

Discitis/ Osteomyelitis:

Infection of the disc and infection of the vertebral body nearly always go together. The reason has to do with the route of seeding; which typically involves seeding of the vertebral endplate (which is vascular), with subsequent eruption and crossing into the disc space, and eventual involvement of the adjacent vertebral body.



In adults, the source is usually from a recent surgery, procedure, or systemic infection. In children it's usually from hematogenous spread. For the Step 1 trivia: Staph A is the most common bug, and think gram negatives with an IV drug user. Almost always (80% of the time) the ESR and CRP are elevated.

Imaging: Early on it's very hard to see with plain films, you will need MRI. You are looking for paraspinal and epidural inflammation, T2 bright disc signal, and disc enhancement. Remember Gallium is superior to WBC scan in the spine. *This is discussed in the neuro chapter - spine section.*

Pott's Disease: TB of the spine is more common in "developing" countries. It behaves in a few different ways, and that makes it easy to test on.

Things to know about TB in the spine:

- * *It tends to spare the disc space*
- * *It tends to have multi-level thoracic "skip" involvement*
- * *Buzzword "Calcified Psoas Abscess"*
- * *Buzzword "Gibbus Deformity" — which is a destructive focal kyphosis*

Septic Arthritis You see this the most in large joints which have an abundant blood supply to the metaphysis (shoulder, hip, knee). *IV drug users will get it in the SI joint, and sternoclavicular joint.* Conventional risk factors include being old, having AIDS, RA, and prosthetic joints. On plain film you might see a joint effusion, or MRI will show synovial enhancement. If untreated this will jack your joint in less than 48hours.

Pneumoarthrogram Sign - If you can demonstrate air within a joint - you can exclude a joint effusion. No joint effusion = No septic joint.

Necrotizing Fasciitis: This is a very bad actor that kills very quickly. The good news is that it's pretty rare, typically only seen in HIVers, Transplant patients, diabetics, and alcoholics. It's usually polymicrobial (the second form is Group A Strep). **Gas is only seen in a minority of cases, but if you see gas in soft tissue this is what they want.** Diffuse fascial enhancement is what you'd see if the ER is dumb enough to order cross sectional imaging (they often are). Fournier Gangrene is what they call it in the scrotum.

TB: This is a special topic (high yield) with regard to MSK infection. It's not that common, with <5% of patients with TB having MSK involvement. Although on multiple choice tests, I think you'll find it appears with a high frequency.

Key Points to know:

- * The vertebral body is involved with sparing of the disc space until late in the disease (very different than more common bacterial infections).
- * *"Gibbus Deformity"* is a focal kyphosis seen in *"Potts Disease"*, among many other things.
- *"Rice Bodies"* - These are sloughed, infarcted synovium seen with end stage RA, and TB infection of joints.
- *Tuberculosis Dactylitis (Spina Ventosa)* - Typically affects kids more than adults with involvement of the short tubular bones of the hands and feet. It is often a smoldering infection without periosteal reaction. Classic look is a **diaphyseal expansile lesion** with soft tissue swelling.

Aggressive Lesions

There are tons of primary osseous malignancies, the most common are myeloma/plasmacytoma (27%), Osteosarcoma (20%) and Chondrosarcoma (20%). According to Helms, the **wide zone of transition is the best sign that a lesion is aggressive**. This is actually a useful pearl.

Myeloma /Plasmacytoma /Mets - Discussed in the cystic bone lesion section

Osteosarcoma: There are a bunch of subtypes, but for the purpose of this discussion there are 4. Conventional Intramedullary (85%), Parosteal (4%), Periosteal (1%), Telangiectatic (rare). All the subtypes produce bone or osteoid from neoplastic cells. Most are idiopathic but you can have secondary causes *{usually seen in elderly}* XRT, Pagets, Infarcts, etc...

Conventional Intramedullary': More common, and higher grade than the surface subtypes (periosteal, and parosteal). Primary subtypes typically occur in young patients (10-20). The most common location is the femur (40%), and proximal tibia (15%).

Buzzwords include various types of aggressive periosteal reactions:

- * “Sunburst”- periosteal reaction that is aggressive and looks like a sunburst
- * *Codman triangle* - With aggressive lesions, the periosteum does not have time to ossify completely with new bone (e.g. as seen in single layer and multi-layered periosteal reaction), so only the edge of the raised periosteum will ossify - creating the appearance of a triangle.
- * *Lamellated* (onion skin reaction) - multi layers of parallel periosteum, looks like an onion’s skin.

High Yield Trivia:

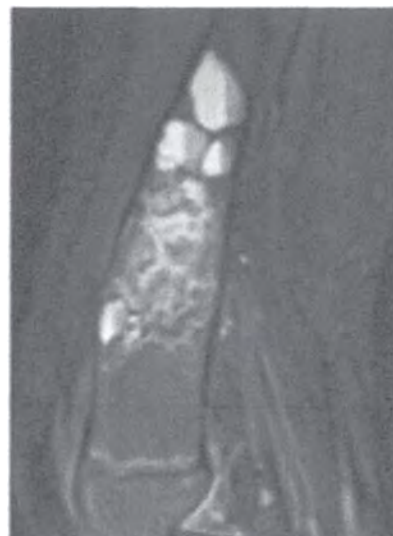
- * *Osteosarcoma met to the lung is a “classic ” (frequently tested) cause of occult pneumothorax.*
- * **“Reverse Zoning Phenomenon”** - more dense mature matrix in the center, less peripherally *{opposite of myositis ossificans}*.

Parosteal Osteosarcoma: Generally low grade, BULKY parosteal bone formation. Think Big... just say Big. This guy loves the posterior distal femur (*because of this location it can mimic a cortical desmoid early on*). The lesion is metaphyseal 90% of the time. The buzzword is “*string sign*” - which refers to a radiolucent line separating the bulky tumor from the cortex.

Periosteal Osteosarcoma
Worse prognosis than parosteal but better than conventional osteosarcoma.
Tends to occur in the diaphyseal regions, classic medial distal femur.

<i>This vs That: Parosteal vs Periosteal Osteosarcoma</i>	
Parosteal	Periosteal
Early Adult / Middle Age	Age Group (15-25)
Metaphysis (90%)	Diaphyseal
Likes Posterior Distal Femur	Likes Medial Distal Femur
Marrow extension (50%)	Usually no marrow extension
Low Grade	Intermediate Grade

Telangiectatic Osteosarcoma: About 15% have a narrow zone of transition. Fluid-Fluid levels on MRI is classic. They are High on T1 (from methemoglobin). Can be differentiated from ABC or GCT (maybe) by tumor nodularity and enhancement.



Fluid-Fluid Levels-ODx:

- Telangiectatic Osteosarcoma
- Aneurysmal Bone Cyst
- Giant Cell Tumor

Chondrosarcoma: Usually seen in older adults (M>F). Likes flat bones, limb girdles, proximal tubular bones. Can be central (intramedullary) or peripheral (at the end of an osteochondroma). Most are low grade.

Risk Factors: Pagets, and anything cartilaginous (osteochondromas, maffucci’s etc...)

If you want to say chondroblastoma but it’s an adult think clear cell chondrosarcoma

Ewings: *Permeative lesion in the diaphysis of a child = Ewings (could also be infection, or EG). Extremely rare in African-Americans. Likes to met bone to bone. Does NOT form osteoid from tumor cells, but can mimic osteosarcoma because of its marked sclerosis (sclerosis occurs in the bone only, not in the soft tissue — which is NOT the case in osteosarcoma).*

Chordoma Usually seen in adults (30-60), usually slightly younger in the clivus and slightly older in the sacrum. Most likely questions regarding the chordoma include location (most common sacrum, second most common clivus, third most common vertebral body), and the fact that they are very T2 bright.

Chordoma Most Commons:

- * Most common primary malignancy of the spine.
- * Most common primary malignancy of the sacrum.
- * When involving the spine, most common at C2.

<i>Clivus Chordomas are midline, Chondrosarcomas are off midline.</i>

Aggressive Soft Tissue Lesions

Fibrosarcoma /Malignant Fibrous Histiocytoma (MFH)

- Fibrosarcoma: Just like osteosarcoma can be primary or secondary (from Pagets, infarct etc..) These are lytic malignant tumors that DO NOT produce osteoid or chondroid matrix. They are almost “Always Lytic”, and may be permeative or moth eaten. Also “NOT T2 Bright” - which most tumors are.
- MFH: This actually used to be lumped in with Fibrosarcoma - but now they are separate. MFHS are way more common than Fibrosarcomas (*most common soft tissue sarcoma in adults*). From a radiology perspective, they look the same. So when you say one you should say the other.

Trivia: Bone infarcts can turn into MFH - “sarcomatous transformation of infarct”

Synovial Sarcoma: Seen most commonly in the lower extremities of patients aged 20-40. They occur close to the joint (but not in the joint). To confuse the issue they may have secondary invasion into the joint (10%), however for the purpose of multiple choice tests they “never involve the joint.”

They could show this tumor in 3 different ways: (1) as the “triple sign”, which is high, medium, and low signal all in the same mass (probably in the knee) on T2, (2) as the “bowl of grapes” which is a bunch of fluid-fluid levels in a mass (probably in the knee), or (3) as a plain x-ray with a soft tissue component and calcifications - this would be the least likely way to show it.

Synovial Sarcoma Trivia:

- * Most sarcomas don't attack bones; Synovial Sarcoma Can
- * Most sarcomas present as painless mass; Synovial Sarcomas Hurt
- * Soft tissue calcifications + Bone Erosions are highly suggestive
- * They are slow growing and small in size often leading to people thinking they are B9.
- * 90% have a translocation of X-18.
- * Most common malignancy of the foot, ankle, and lower extremity

Liposarcoma - This is the second most common soft tissue sarcoma. You see it in middle aged people (40-60), with the classic location being the retroperitoneum (can also happen in the extremities). The most common type (well-differentiated) is also the least aggressive.

When I say “Fatty Mass in the retroperitoneum, ”you say Liposarcoma

Things that make you think it is a liposarcoma (and not a lipoma)

- Inhomogenous attenuation - soft tissues masses in the fat
- Infiltration of adjacent structures

Lymphoma - Discussed in the peds chapter.

Treatment High Yields

- *Osteosarcoma:* Chemo first (to kill micro mets), followed by wide excision
- *Ewings:* Both Chemo and Radiation, followed by wide excision.
- *Chondrosarcoma:* usually just wide excision (they are usually low grade, and main concern is local recurrence).
- *Giant Cell Tumor:* Because it extends to the articular surface usually requires arthroplasty.

“Don’t Touch Lesions” - Characteristically Benign Lesions, that look Aggressive but are NOT - and should NOT be biopsied because of possibly misleading pathology.		
Myositis Ossificans	Circumferential calcifications with a lucent center	Can look scary on MRI if imaged early because of edema, and avid enhancement
Avulsion Injury	Typical location near the pelvis	Can have an aggressive periosteal reaction
Cortical Desmoid	Characteristic location on the posterior medial epicondyle of the distal femur	Can be hot on bone scan.
Synovial Herniation Pit “Pitt’s Pit”	Characteristic location in the femoral neck	Lytic appearing lesion

Bone Biopsy - The route of biopsy should be discussed with the orthopedic surgeon, to avoid contaminating compartments not involved by the tumor (or not going to be used in the resection process).

Special considerations:

- Pelvis: Avoid crossing gluteal muscles (may be needed for reconstruction).
- Knee: Avoid the joint space via crossing suprapatellar bursa or other communicating bursae. Avoid crossing the quadriceps tendon unless it is involved.
- Shoulder: Avoid the posterior 2/3rd (axillary nerve courses post -> anterior, therefore a posterior resection will denervate the anterior 1/3).

B9 Lesions

FEGNOMASHIC is the mnemonic for cystic bone lesions made popular by Clyde Helms. As it turns out, you can rearrange the letters of FEGNOMASHIC to form a word FOGMACHINES. I find it a lot easier to remember a mnemonic if it actually forms a real word. Having said that the whole idea of memorizing a list of 11 or 12 things is really stupid. You would never give a differential that included all of those, they occur in different places, in different ages, and often look very different. Differentials (for people who know what they are looking at) are usually never deeper than 3 or 4 things. If you are giving a differential of 12 things, just say you don't know what it is.

First a brief discussion of location & Age

Age:

The key to remember is that

- * < 30 = EG, ABC, NOF, Chondroblastoma, and Solitary Bone Cysts
- * Any Age = Infection
- * > 40 = Mets and Myeloma (unless it's neuroblastoma mets).

Epiphysis:

In general, only a few lesions tend to arise in the epiphysis. The "four horseman of the apophysis" is the mnemonic I like to use, and I think about the company AIG that was involved in some scandal a few years ago. AIG "the evil" Company.

Epiphyseal Equivalents:

Big ones to remember are the carpals, the patella, the greater trochanter, and the calcaneus

ABC, Infection, Giant Cell, and Chondroblastoma.

**The caveat is that ABC is usually metaphyseal but after the growth plate closes it can extend into the epiphysis.*

For the purpose of multiple choice tests it is important to not forget about the malignant tumor at the end of the bone (epiphysis) - Clear Cell Chondrosarcoma. This guy is slow growing, with a variable appearance (lytic, calcified, lobulated, ill defined, etc...). Just remember if they say malignant epiphyseal you say Clear Cell Chondrosarcoma.

Metaphysis

The metaphysis is the fastest growing area of a bone, with the best blood supply. This excellent blood supply results in an increased predilection for Mets and Infection. Most of the cystic bone lesions can occur in the metaphysis.

Diaphysis

Just like the metaphysis, most entities can occur in the diaphysis (they just do it less).

Now a discussion on the pathology

Fibrous Dysplasia: Fibrous dysplasia is a skeletal developmental anomaly of osteoblasts - failure of normal maturation and differentiation. The disorder can occur at any age . It can be monostotic (20s & 30s) or polyostotic (< 10 year old).

Famously has a very variably appearance, with phases like pagets (lytic, mixed, blastic). The buzzword is “ground glass.” The catch phrase is “long lesion in a long bone.” The textbook appearance “lytic lesion with a hazy matrix” The discriminator used by Helms is “no periosteal reaction or pain.”

Likes the ribs and long bones. If it occurs in the pelvis, it also hits the ipsilateral femur (Shepherd Crook deformity). If it’s multiple it likes the skull and face (Lion-like faces).

<i>This vs That: McCune Albright vs Mazabraud Syndrome</i>	
McCune Albright	Mazabraud
Polyostotic Fibrous Dysplasia	Polyostotic Fibrous Dysplasia
Girl	Woman {middle aged}
Cafe au lait spots	Soft Tissue Myxomas
Precocious Puberty	Increased Risk Osseous Malignant Transformation



Adamantinoma: A total zebra {probably a unicorn}. A tibial lesion that resembles fibrous dysplasia (mixed lytic and sclerotic). It is potentially malignant.

Enchondroma: This guy is a tumor of the medullary cavity composed of hyaline cartilage. It appears as a lytic lesion with irregularly speckled calcification of chondroid matrix, classically described as **ARCS AND RINGS**. Having said that the **chondroid matrix is not found in the fingers or toes** *** *this is a high yield factoid for the purpose of multiple choice tests*. The enchondroma is actually the most common cystic lesion in the hands and feet. Just like fibrous dysplasia this lesion does not have periostitis.

The trick to differentiating enchondroma vs a low grade chondrosarcoma is the history of pain.

<i>This vs That: Ollier's vs Maffucci's</i>	
Ollier's	Maffucci's
ONLY Enchondromas	MORE than just Enchondromas (also Hemangiomas)
	Malignant potential (20% turn into chondrosarcoma, and other cancers GI, Ovary)

Eosinophilic Granuloma (EG): This is typically included in every differential for people less than 30. It can be solitary (usually) or multiple.

There are 3 classic appearances - for the purpose of multiple choice:

- (1) Vertebra plana in a kid
- (2) Skull with lucent "beveled edge" lesions (also in a kid).
- (3) "Floating Tooth" with lytic lesion in alveolar ridge — this would be a differential case

The appearance is highly variable and can be lytic or blastic, with or without a sclerotic border, and with or without a periosteal response. Can even have an osseous sequestrum.

Classic DDx for Vertebra Plana (MELT)

- * Mets / Myeloma
- * EG
- * Lymphoma
- * Trauma / TB

Classic DDx for Osseous Sequestrum:

- * Osteomyelitis
- * Lymphoma
- * Fibrosarcoma
- * EG
- * * Osteoid Osteoma can mimic a sequestrum

Giant Cell Tumor (GCT): This guy has some key criteria (which lend themselves well to multiple choice tests). They include:

- Physis **MUST** be closed
- Non Sclerotic Border
- Abuts the articular surface

Another trick is to show you a pulmonary met, and ask if it could be GCT? The answer is yes (although this is rare) GCT is considered “quasi-malignant” because they can be locally invasive and about 5% will have pulmonary mets (which are still curable by resection). As a result of this, they should be resected with wide margins.

Things to know about GCTs:

- Most common in the knee - abutting the articular surface
- Most common at age 20-30 * physis must be closed
- There is an association with ABCs (they can turn into them)
- They are “quasi-malignant” - 5% have lung mets

Nonossifying Fibroma (NOF): These are very common. They are seen in children, and will spontaneously regress (becoming more sclerotic before disappearing). They are *rare in children not yet walking*. Just like GCTs they like to occur around the knee. They are classically described as eccentric with a thin sclerotic border (remember GCTs don't have a sclerotic border). They are called fibrous cortical defects when smaller than 2cm.



Jaffe-Campanacci Syndrome: Syndrome of multiple NOFs, cafe-au-lait spots, mental retardation, hypogonadism, and cardiac malformations.

Osteoid Osteoma “*Pain at night, relieved by aspirin.*” It's classically found in two spots (1) meta/diaphysis of long bones and (2) the posterior elements of the spine. One way to test this is to show a plain film that is probably an osteoid osteoma then follow it with an MRI showing “lots of edema.” I'll say that again “*large amount of edema for the size of the lesion.*”

Another piece of trivia is that when you have them in the spine (most common in the lumbar spine), you frequently have an associated **painful scoliosis** with the **convexity pointed away from the lesion**. These can be treated with percutaneous radiofrequency ablation (as long as it's not within 1cm of a nerve or other vital structure - *typically avoided in hands, spine, and pregnant patients*).

Association of Osteoid Osteoma

Painful Scoliosis

Growth Deformity: Increased length and girth of long bones

Synovitis: Can be seen if intra-articular, leading to early onset arthritis

Arthritis: Can occur from primary synovitis, or secondarily from altered joint mechanics.

Osteoblastoma: Basically it's an osteoid osteoma that is larger than 2cm. It's seen in patients < 30years old. They are most likely to show this in the posterior elements. It also occurs in the long bones (35%) and when it does it is usually diaphyseal (75%).

Metastatic Disease: Should be on the differential for any patient over 40 with a lytic lesion.
As a piece of trivia renal cancer is ALWAYS lytic (usually).

Classic Blastic Lesions: Prostate, Carcinoid, Medulloblastoma

Classic Lytic Lesions: Renal and Thyroid

Multiple Myeloma (MM): Plasma cell proliferation increases surrounding osteolytic activity (in case someone asks you the mechanism). Usually in older patient (40s-80s).
Plasmacytomas can precede clinical or hematologic evidence of myeloma by 3 years.

They usually have discrete margins, and can be solitary or multiple. Vertebral body destruction with sparing of the posterior elements is classic. Bone Scan is often negative, *skeletal survey is better* (but horrible pain to read), and MRI is the most sensitive.

Additional classic (testable) scenario: *MM manifesting as Diffuse Osteopenia*

Myeloma Related Conditions:

Plasmacytoma (usually under 40): This is a discrete, solitary mass of neoplastic monoclonal plasma cells in either bone or soft tissue (*extramedullary sub type*). It is associated with latent systemic disease in the majority of affected patients. It can be considered as a singular counterpart multiple myeloma. The lesions look like a geographic lytic area, sometimes with expansile remodeling.

"Mini Brain Appearance " - Plasmacytoma in vertebral body

POEMS: This is basically "*Myeloma with Sclerotic Met s*" It's a rare medical syndrome with plasma cell proliferation (typically myeloma) with neuropathy, and organomegaly.

Aneurysmal Bone Cyst (ABC): Aneurysmal bone cysts are aneurysmal lesions of bone with thin-walled, blood-filled spaces (fluid-fluid level on MRI). Patients are usually < 30. They may develop following trauma.

Location: Tibia > Vert > Femur > Humerus

They can be described as primary ABC, presumably arising de novo or secondary ABC, associated with another tumor (classic GCT). They are commonly associated with other benign lesions.

Classic DDx for Lucent Lesion in Posterior Elements

- * Osteoblastoma
- * ABC
- * TB

Things to know about ABC:

- Up to 40% of secondary ABC's are associated with giant cell tumor of bone.
- It's on the DDx for Fluid - Fluid Level on MRI
- Patient <30
- Tibia is the most common site

Solitary (Unicameral) Bone Cyst.* It would be unusual to see one of these in a patient older than 30. Most common in the tubular bones (90-95)% usually humerus or femur. Unique feature: "Always located centrally."

It's going to be shown one of two ways: (1) With a fracture through it in the humerus (probably with a fallen fragment sign) or (2) As a lucent lesion in the calcaneus (probably with a fallen fragment sign).

The fallen fragment sign (Tone fragment in the dependent portion of a lucent bone lesion) is pathognomonic of solitary bone cyst.

Brown Tumor (Hyperparathyroidism): The "brown tumor" represents localized accumulations of giant cells and fibrous tissue (in case someone asks). They appear as lytic or sclerotic lesions with other findings of hyperparathyroidism (subperiosteal bone resorption). In other words, they need to tell you he/she has hyperparathyroidism first. They may just straight up tell you, or they will show you some bone resorption first (classically on the side of a finger, edge of a clavicle, or under a rib).

Chondroblastoma: This is seen in kids (90% age 5-25). They classically show it in two ways (1) In the epiphysis of the tibia on a 15 year old, or (2) in an epiphyseal equivalent.

So what are the epiphyseal equivalents???

- * *Patella*
- * *Calcaneus*
- * *Carpal Bones*
- * *And all the Apophyses (greater and less trochanter, tuberosities, etc...)*

Features of the tumor include; A thin sclerotic rim, extension across the physeal plate (25-50%), periostitis (30%). Actual location: femur > humerus > tibia . This may show bone marrow edema, and soft tissue edema on MRI (MRI can mislead you into thinking it's a bad thing). This is one of the only bone lesions that is often NOT T2 bright. They tend to reoccur after resection (like 30% of the time).

Gamesmanship Hip: When you have a chondroblastoma in the hip, it tends to *favor the greater trochanter (more than the femoral epiphysis)*.

Chondromyxoid fibroma: This is the least common benign lesion of cartilage. It is usually in patients younger than 30. The typical appearance is osteolytic, elongated in shape, eccentrically located, metaphyseal lesion, with cortical expansion and a “bite” like configuration. Sorta looks like a NOF.

The Hip

Greater Trochanter - Remember this is also an *epiphyseal equivalent* and the chondroblastomas prefer it to the femoral epiphysis. You can get all the other DDxs (ABC, Infection, GCT here as well). Plus, you can have avulsions of the gluteus medius and minimus.

Lesser Trochanter - An avulsion here - without significant clinical history should make you think pathologic fracture.

The Intertrochanteric Region: Classic DDx here : Lipoma, Solitary Bone Cyst, and Monostotic Fibrous Dysplasia.

The Calcaneus

There are several classic lesions that can be shown in the calcaneus. There are also several non-classic lesions that can be shown (sneaky things).

The classic 3:

Solitary Bone Cyst: This will have sharp edges. A thick sclerotic edge with a multiloculated appearance is helpful. The “fallen fragment” will be more in the bottom if shown - although fractures in the calcaneus are much less common than in the arm.



Pseudo-cyst - This is a variation on the normal trabecular pattern, which creates a central triangular radiolucent area. Supposedly the persistence of thin trabeculae, and visible nutrient foramen, along with the classic location are helpful in telling it from the other benign entities.

Interosseous Lipoma: If they show you this, it will either have to have (a) fat density on CT or MRI, or (b) a **central fragment** - stuck within the middle of the fat. This calcification / fat necrosis occurs about 50% of the time in the real world

Sneaky things:

Just remember that the calcaneus is an *epiphyseal equivalent* so **ABC, Infection, GCT, and Chondroblastoma** can all occur there - think about these when the lesion is more posterior.

In the setting of subtalar degenerative change you can get a **geode** that mimics a cystic lesion (think about this in older patients - 60s with obvious arthritis).

Some Random Benign Lesion Differentials

No Periostitis or Pain

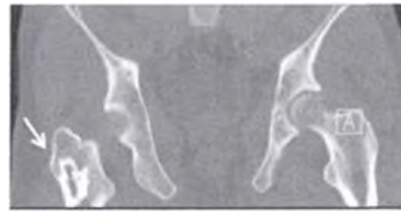
- Fibrous Dysplasia
- Enchondroma
- NF
- Solitary Bone Cyst (unless fractured)

Multiple (FEEMHI)

- * Fibrous Dysplasia
- EG
- * Enchondroma
- # Mets / Myeloma
- * Hyperparathyroidism

Misc Conditions:

Liposclerosing Myxofibroma: *Very characteristic location - at the intertrochanteric region of the femur.* Looks like a geographic lytic lesion with a sclerotic margin. Despite non-aggressive appearance, 10% undergo malignant degeneration so they need to be followed.



LSMF - Classic Location

Osteochondroma: Some people think of this as more of a developmental anomaly (although they still always make the tumor chapter). Actually, it's usually listed as the most common benign tumor. They can be radiation induced, making them the *only benign skeletal tumor associated with radiation*.

They have a very small risk of malignant transformation (which supposedly can be estimated based on size of cartilage cap). Supposedly a cap >1.5cm is concerning.

Key Points:

- * They point away from the joint
- * The bone marrow flows freely into the lesion

Multiple Hereditary Exostosis: AD condition with multiple osteochondromas. They have an **increased risk of malignant transformation**.

Trevor Disease (Dysplasia Epiphysealis Hemimelica - DEH): Osteochondromas develop in an epiphyses causing significant joint deformity (**most common in ankle and knee**). You see this in young children. The osteochondroma looks more like an irregular mass. They tend to be treated with surgical excision.

Supracondylar Spur (Avian Spur): This is an Aunt Minnie, and normal variant. This is an osseous process, that usually does nothing, but can compress the median nerve if the **Ligament of Struthers** smashes it.

This vs That: Osteochondroma vs Supracondylar Spur	
Osteochondroma	Supracondylar Spur
Points AWAY from joint	Points TOWARD the joint

Periosteal Chondroma (Juxta-Cortical Chondroma): When you see a lesion in the finger of a kid think this. It's a rare entity, of cartilaginous origin. "Saucerization" of the adjacent cortex with sclerotic periosteal reaction can be seen.

Osteofibrous Dysplasia: This is a benign lesion found exclusively in the tibia or fibula in children (10 and under - usually). It looks like an NOF , but centered in the anterior tibia, with associated anterior tibial bowing. It can occur with Adamantinoma, and the two cannot be differentiated with imaging.

When I say looks like NOF in the anterior tibia with anterior bowing, you say Osteofibrous Dysplasia.

Tibial Bowing

Most likely shown as an Aunt Minnie - NF-1 anterior with a fibular pseudoarthrosis, Rickets with wide growth plates, or Blounts tibia vara.

The most likely pure trivia question is that physiology bowing is smooth, lateral, and occurs from 18months - 2 years.

NF-1	Anterior Lateral - Unilateral	May be unilateral. May have hypoplastic fibula with pseudoarthrosis.
Foot Deformities	Posterior	
Physiologic Bowing	Lateral - Bilateral Symmetric	Self limiting between 18 months and 2 years.
Hypophosphatasia	Lateral	“Rickets in a newborn”
Rickets	Lateral	Widening and irregularity of the growth plates.
Blount	Tibial Vara - Often asymmetric	Early walking, Fat, black kid.
Osteogenesis Imperfecta	Involves all long bones	
Dwarfs	Short Limbs	

Arthritis

Arthritis is tricky. Anne Brower wrote a book *called Arthritis in Black and White*, which is probably the best book on the subject. The problem is that book is 415 pages. So, I'm going to try and offer the 10 page version.

Epidemiology

Although there are over 90 different rheumatic diseases recognized by the American College of Rheumatology, only a few tend to show up on multiple choice tests (and at the view box).

You can broadly categorize arthritis into 3 categories:

- * Degenerative (OA, Neuropathic)
- * Inflammatory (RA, and Variants)
- * Metabolic (Gout, CPPD)

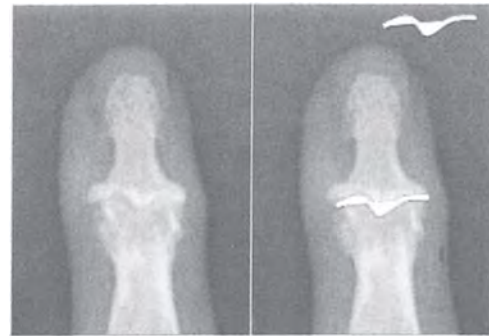
Degenerative:

Osteoarthritis is the most common cause. The pathogenesis is that you have mechanical breakdown (hard work) which leads to cartilage degeneration (fissures, microfractures) and fragmentation of subchondral bone (sclerosis, and subchondral cysts). You get all the classic stuff, joint space narrowing fNOT symmetric^ subchondral cysts, endplate changes, vacuum phenomenon, etc... The poster boy is the osteophyte.

Neuropathic Joint. The way the case is classically shown is a bad joint followed by the reason for a bad joint (syringomyelia, spinal cord injury, etc...). A way to think about this is "*osteoarthritis with a vengeance*." The buzzword is "Surgical Like Margins." Basically nothing else causes this kind of destruction. I like to describe the joints as a deformity, with debris, and dislocation, having dense subchondral bone, and destruction of the articular cortex. The classic scenario is a shoulder that looks like it's been amputated, and then they show you a syrx.

Inflammatory:

Erosive Osteoarthritis (*Inflammatory Osteoarthritis*). The buzzword is “gull wing”, which describes the central erosions. It is seen in postmenopausal women and favors the DIP joints.

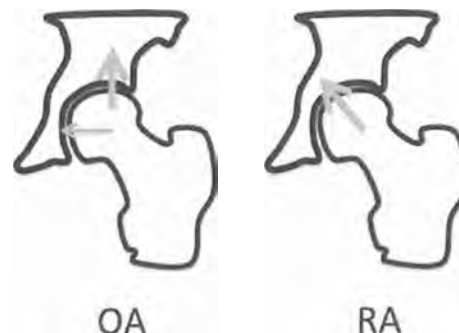


Erosive OA - Gullwing

Rheumatoid Arthritis: There is a ton of trivia related to this disease. It's not a disease of bone production. Instead it is characterized by osteoporosis, soft tissue swelling, marginal erosions and uniform joint space narrowing. It's often bilateral and symmetric. Classically spares the DIP joints (opposite of erosive OA).

- * **Felty Syndrome:** RA > 10 years + Splenomegaly + Neutropenia
- * Caplan Syndrome: RA + Pneumoconiosis

The distribution of RA vs OA in the hip is a classic teaching point:



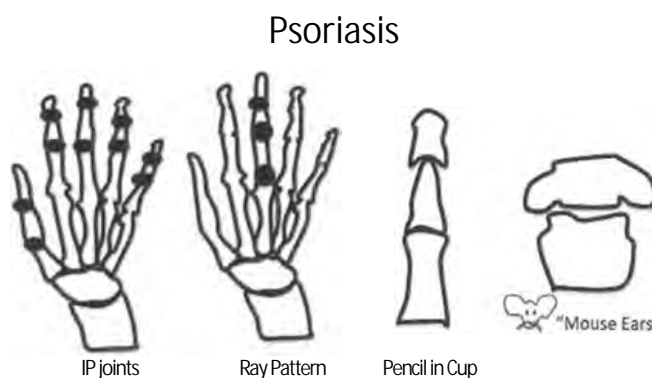
Rheumatoid Variants

- * Psoriasis
- * Reiter's syndrome (*Reactive arthritis*)
- * Ankylosing Spondylitis
- * Inflammatory Bowel Disease

Psoriasis: This is seen in 30% of patients with psoriasis. In almost all cases (90%) the skin findings come first, then you get the arthritis. As a point of trivia, there is a strong correlation between involvement of the nail and involvement of the DIP joint. The classic description is “erosive change with bone proliferation (IP joints > MCP joints). The erosions start in the margins of the joint and progress to involve the central portions (can lead to a “pencil sharpening” effect). The hands are the most commonly affected (second most common is the feet). Up to 40% of cases will have SI joint involvement (asymmetric).

Additional Buzzwords

- * “Fuzzy Appearance” to the bone around the joint (bone proliferation)
- * Sausage Digit - whole digit has soft tissue swelling
- * Ivory Phalanx - sclerosis and/or bone proliferation (most commonly the great toe)
- * Pencil in Cup Deformities
- * Ankylosis in Finger
- * “Mouse Ears”



When I say Ankylosis in the Hand, You Say (1) Erosive OA or (2) Psoriasis

RA	Psoriasis
Symmetric	Asymmetric
Proximal (favors MCP, carpals)	Distal (favors IP joints)
Osteoporosis	No Osteoporosis
No Bone Proliferation	Bone Proliferation
Can Cause “Mutilans” When Severe	Can Cause “Mutilans” When Severe

Reiter's (Reactive arthritis): Apparently Reiter was a Nazi (killed a bunch of people with typhus vaccine experiments). So, people try not to give him any credit for things (hence the name change to Reactive arthritis). Regardless of what you call it, it's **a very similar situation to Psoriatic arthritis** - both have bone proliferation and erosions, and asymmetric SI joint involvement. The difference is that **Reiter's is rare in the hands** (tends to affect the feet more). Just remember Reiters below the waist.

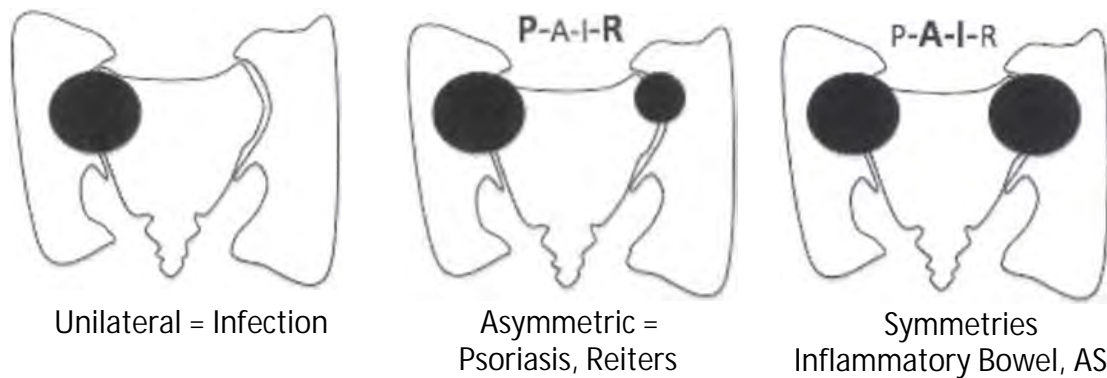
Ankylosing Spondylitis: This disease favors the spine and SI joints. The classic buzzword is **"bamboo spine"** from the syndesmophytes flowing from adjacent vertebral bodies. Shiny corners is a buzzword, for early involvement. As you might imagine these spines are susceptible to fracture in trauma. **SI joint involvement is usually the first site (symmetric)**. The joint actually widens a little before it narrows. As a point of trivia, these guys can have an upper lobe predominant interstitial lung disease, with small cystic spaces.

Random High Yield Topic: Ankylosing Spondylitis in the Hip
When the peripheral skeleton is involved in patient's with Ank Spond, think about the shoulders and hips (hips more common). Hip involvement can be very disabling.
Heterotopic Ossification tends to occur post hip replacement or revision. It occurs so much that they often get postoperative low dose radiation and NSAIDs to try as prophylactic therapy.
If they show you normal SI joints - then show you anything in the spine it's not AS. It has to hit the SI joints first (especially on multiple choice).

Key Point with regard to Sacroiliitis: The hyaline cartilage is thinner at the ilium, and thicker at the sacrum. **So erosions occur at the iliac side first.**

Inflammatory Bowel Disease - This occurs in two distinct flavors.

- (A) : Axial Arthritis (favors SI joints and spine) - often unrelated to bowel disease
- (B) : Peripheral Arthritis - this one varies depending on the severity of the bowel disease.



Psoriatic Arthritis	Reiters (Reactive)	Ankylosing Spondylitis
M = F	M > F	M>F
Asymmetric SI Joint	Asymmetric SI Joint	Symmetric SI Joint
Hands, Feet, Thoracolumbar Spine	Feet, Lumbar Spine, SI joint	SI joint, Spine (whole thing)

Metabolic:

Gout: This is a crystal arthropathy from the deposition of uric acid crystals in and around the joints. It's almost always in a man over 40. The big toe is the classic location.

Buzzwords / Things to Know:

- * Earliest Sign = Joint Effusion
- * Spares the Joint Space (until late in the disease)
- * "Punched out lytic lesions"
- * "Overhanging Edges"
- * Soft tissue tophi

Gout Mimickers: There are 5 entities that can give a similar appearance to a gouty arthritis, although they are much less common. The way I remember them is

"American Roentgen Ray Society Hooray"

- * *Amyloid*
- * *RA (cystic)*
- * *Reticular Histocytosis (the most rare)*
- * *Sarcoid*
- * *Hyperlipidemia*

CPPD: Calcium Pyrophosphate Dihydrate Disease is super common in old people. It often causes chondrocalcinosis (although there are other causes). Synovitis + CPPD = “Pseudogout.” CPPD loves the triangular fibrocartilage of the wrist, the peri odontoid tissue, and intervertebral disks. Another important phrase is “degenerative change in an uncommon joint” - shoulder, elbow, patellofemoral joint, radiocarpal joint. Having said that pyrophosphate arthropathy is most common at the knee.

- * *If you see isolated disease in the patellofemoral, radiocarpal, or talonavicular joint think CPPD.*
- * *Hooked MCP Osteophytes with chondrocalcinosis in the TFCC is a classic look (although hemochromatosis can also look that way).*

“Milwaukee Shoulder” This is a destruction of the shoulder (almost looks neuropathic) but is secondary to hydroxyapatite. The articular surface changes will be very advanced, and you have a lot of intra-articular loose bodies. It’s classically seen in an old woman with a history of trauma to that joint.

OA vs CPPD? There are many overlapping features including joint space narrowing, subchondral sclerosis, subchondral cyst, and osteophyte formation. However, CPPD has some unique features such as an “atypical joint distribution” - favoring compartments like the patellofemoral or radiocarpal. Subchondral cyst formation can be bigger than expected.

Hemochromatosis - This iron overload disease also is known for calcium pyrophosphate deposition and resulting chondrocalcinosis. It has a similar distribution to CPPD (MCP joints). Both CPPD and Hemochromatosis will have “hooked osteophytes” at the MCP joint. As a point of trivia, therapy for the systemic disease does NOT affect the arthritis.

Hyperparathyroidism - As you may remember from medical school this can be primary or secondary, and its effects on calcium metabolism typically manifest in the bones. Here are your buzzwords: “Subperiosteal bone resorption” of the radial aspect 2nd and 3rd fingers, rugger-jersey spine, brown tumors, tenninal tuft erosions.

The classic ways this is shown:

- * *Superior and inferior rib notching - bone resorption*
- * *Resorption along the radial aspect of the fingers with brown tumors*
- * *Tuft Resorption*
- * *Rugger Jersey Spine*
- * *Pelvis with Narrowing or “Constricting” of the femoral necks, and wide SI joints.*

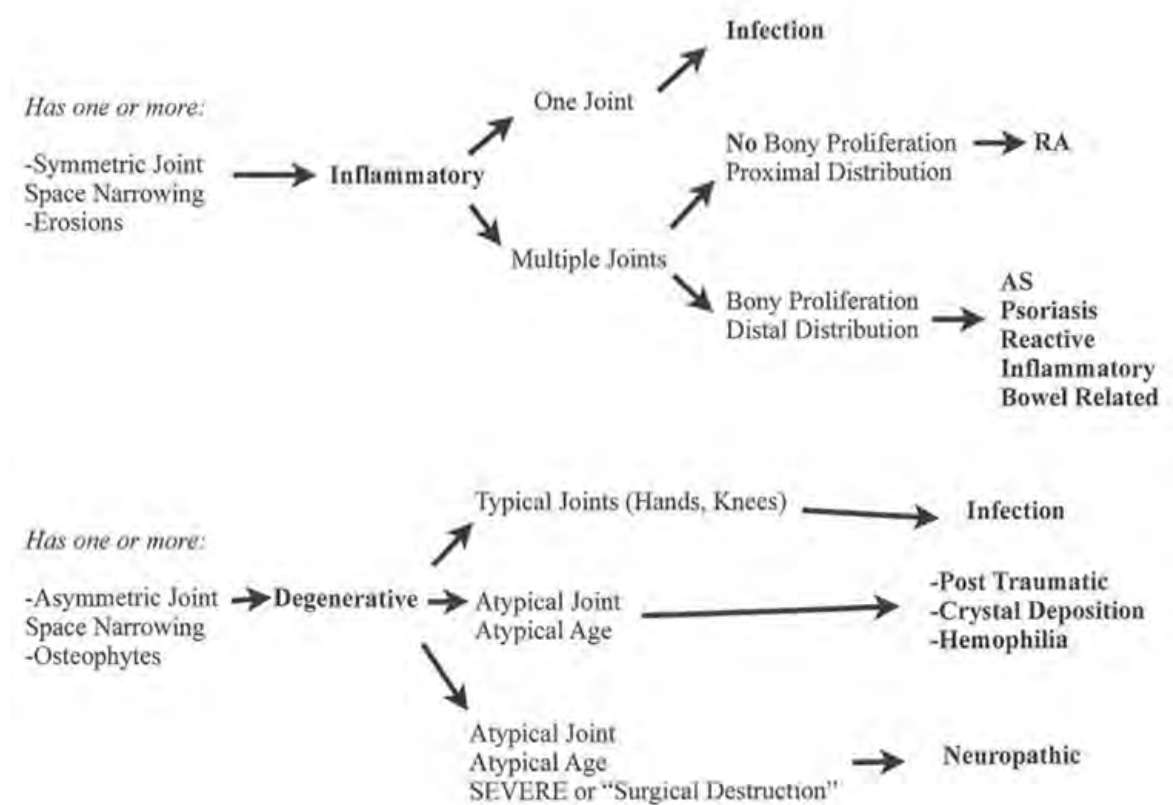
Hyperparathyroidism



Subperiosteal Resorption, Tuft Resorption
and brown tumors

Rugger Jersey Spine Brown Tumor

Problem Solving: If you are given a picture of a hand or foot and asked what the arthritis is, it will probably be obvious (they show a gullwing for erosive OA, or bad carpals for RA, or the pencil in cup for psoriasis, or the 5th metatarsal for RA). If it's not made obvious with an "Aunt Minnie" appearance I like to use this approach to figure it out (I also use this in the real world).



Spine Degenerative Change: In the real world it's usually just multilevel degenerative change. But in multiple choice world you should be thinking about other things. Shiny corners with early AS, or flowing syndesmophytes with later AS. DISH with the bulky osteophytes sparing the disc space. The big bridging lateral osteophyte is classically shown for psoriatic arthritis.

Vertebral Osteophytes	
"Flowing Syndesmophytes"	Ankylosing Spondylitis
Diffuse Paravertebral Ossifications	DISH
Focal Lateral Paravertebral Ossification	Psoriatic Arthritis

Cervical Spine: *Gamesmanship*

- * **Fusion:** Either congenital (Klippel-Feil) or Juvenile RA.
- * **Erosions of the Dens:** CPPD and RA famously do this.
- * **Bad Kyphosis** = NF1

Misc Stuff That's Sorta in the Arthritis Category:

Systemic Lupus Erythematosus: The Aunt Minnie Look is **reducible deformity of joints without articular erosions**. *Joint space narrowing and erosions are uncommon findings.* They can show you the hands with ulnar subluxations at the MCPs on Norgaard view, then they reduce on AP (because the hands are flat).

This ligamentous laxity also increases risk of **patellar dislocations**.



SLE: Shows Reversible Ulnar Deviation

Jaccoud's Arthropathy: This is **very similar to SLE** in the hand (people often say them together). You have non erosive arthropathy with ulnar deviation of the 2nd-5th fingers at the MCP joint. The **history is post rheumatic fever**.

DISH (Diffuse Idiopathic Skeletal Hyperostosis) : You see ossification of the anterior longitudinal ligament involving more than 4 levels with **sparing of the disc spaces**, you say DISH. The **thoracic spine is most commonly used**. These guys often have bony proliferation at pelvis, ischial tuberosities, at the trochanters, and iliac crests. There is **no sacroiliitis** (helps you differentiate from AS).

OPPL (Ossification of the Posterior Longitudinal Ligament): This is an ossification of the posterior longitudinal ligament. It is associated with DISH, ossification of the ligamentum flavum, and Ankylosing Spondylitis. It favors the cervical spine of old Asian men. It **can cause spinal canal stenosis, and lead to cord injury after minor trauma**. A key point is that it's bad news in the cervical spine (where it is most common), in the thoracic spine it is usually asymptomatic.

Destructive Spondyloarthropathy.: This is associated with patients on renal dialysis (for at least 2 years), and it most commonly affects the C-spine. It looks like bad degenerative changes or CPPD. Amyloid deposition is supposedly why it happens.

Mixed Connective Tissue Disease: One unique feature is that it is positive for some antibody - Ribonucleoprotein (RNP), and therefore *serology is essential to the diagnosis*.

Juvenile Idiopathic Arthritis: This occurs before age 16 (by definition). What you see is a washed out hand that has a proximal distribution (**carpals are jacked**), and ankylosed (**premature fusion of growth plates**). Serology is often negative (85%). In the knees, you see enlargement of the epiphyses and widened intercondylar notch - similar to findings in hemophilia.



JRA

- Note the effect on the carpals

Amyloid Arthropathy: This is seen with patients on dialysis (less commonly in patients with chronic inflammation such as RA). The pattern of destruction can be severe - similar to septic arthritis or neuropathic spondyloarthropathy. The distribution is key, the **bilateral involvement of the shoulders, hips, carpals, and knees** being typical. **Carpal tunnel syndrome is a common clinical manifestation**. The **joint space is typically preserved** until later in the disease. When associated with dialysis it's rare before 5 years of treatment, but very common after 10 years (80%).

Congenital

Dwarfs, Coalitions, and Feet are discussed in detail in the PEDs chapter

Total Hip Arthroplasty Complications:

Loosening: This is the most common indication for revision. The criteria on x-ray is **>2mm at the interface** (suggestive). If you see **migration of the component** you can call it (*migration includes varus tilting of the femoral stem*).

Particle Disease: Any component of the device that sheds will cause an inflammatory response. Macrophages will try and eat the particles and spew enzymes all over the place.

Things to know about particle disease (in THA):

- * Most commonly seen in non-cemented hips
- * Tends to occur 1-5 years after surgery
- * X-ray shows “smooth” endosteal scalloping (distinguishes from infection)
- * Produces no secondary bone response
- * Can be seen around screw holes (particles are transmitted around screws)

Stress Shielding: The stress is transferred through the metallic stem, so the bone around it is not loaded. Orthopods call this “Wolff’s Law” - where the unloaded bone just gets resorbed. The trivia to know is that it (1) happens more with uncemented arthroplasty and (2) increases the risk of fracture.

Wear Patterns: It is normal to have a little bit of thinning in the area of weight bearing - this is called “Creep.” It is not normal to see wear along the superior lateral aspect.

Polyethylene Wear	Creep
Superior - Lateral	Axial Direction
Pathologic	Normal

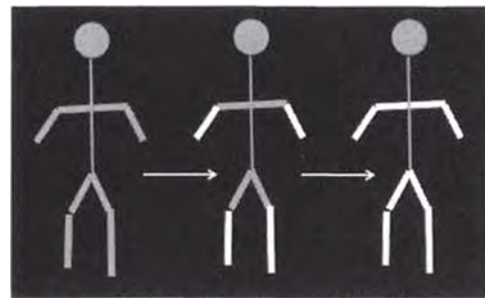
Heterotopic Ossifications: This is very common (15-50%). It’s usually asymptomatic. The trivia regarding multiple choice tests is that “hip stiffness” is the most common complaint. Also in Ank Spon patients, because they are so prone to heterotopic ossifications, they sometimes give them low dose prophylactic radiation prior to THA.

Marrow

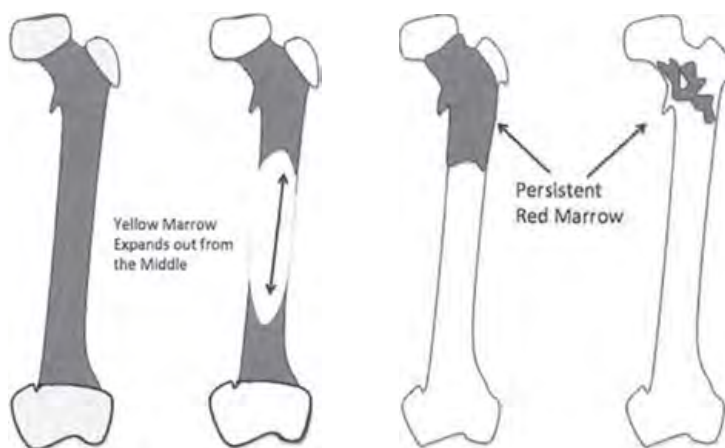
This is a confusing topic and there are entire books on the subject. I'm going to attempt to hit the main points, and simplify the subject.

Bone marrow consists of three components: (1) Trabecular Bone - the support structure, (2) Red Marrow - for making blood, and (3) Yellow Marrow -fat for a purpose unknown at this time.

Marrow Conversion: The basic rules are that yellow marrow increases with age, in a predictable and progressive way. This is usually completed by the mid 20s. You are born with all red marrow, and the conversion of red to yellow occurs from the extremities to the axial skeleton (feet and hands first). Within each long bone the progression occurs epiphyses / apophyses first -> diaphysis -> followed by the distal metaphysis, and finally the proximal metaphysis. **Red marrow can be found in the humeral heads and femoral heads as a normal variant in adults.**



Red Marrow Converts to Yellow Marrow from Distal to Proximal



Baby

Child Teenager Adult

So as a child you have diffuse red marrow except for ossified epiphyses and apophyses. As an adult you have yellow marrow everywhere except in the axial skeleton, and proximal metaphyses of proximal long bones.

Few Pearls on Marrow:

- Yellow marrow increases with age (as trabecular bone decreases with osteoporosis, yellow marrow replaces it).
- T1 is your money sequence: Yellow is bright, Red is darker than yellow (slightly higher than muscle).
- Red marrow should never be darker than a normal disk or muscle on T1
- Red marrow increases if there is a need for more hematopoiesis (reconversion - occurs in exact reverse order of normal conversion)
- Marrow turns yellow with stress / degenerative change in the spine

This question can be asked in 3 main ways:

- (1) What is the normal pattern of conversion?
- (2) What is the normal pattern of reconversion?
- (3) What areas are spared / normal variants?

(1) The epiphyses convert to fatty marrow almost immediately after ossification. Distal then proceeds medial (diaphysis first, then metaphysis).

(2) The pattern of reconversion: This occurs in the reverse order of normal marrow conversion, beginning in the axial skeleton and heading peripheral. The last to go are the more distal long bones. Typically the epiphyses are spared unless the hematopoietic demand is very high.

Spine -> Flat Bones -> Long Bone Metaphysis -> Long Bone Diaphysis -> Long Bone Epiphyses

(3) Patchy areas of red marrow may be seen in the proximal femoral metaphysis of teenagers. The **distal femoral sparring is especially true in teenagers and menstruating women.**

Leukemia: Proliferation of leukemic cells results in replacement of red marrow. **Marrow will look darker than muscle (and normal disks) on T1.** On STIR maybe higher than muscle because of the increased water content. T2 is variable often looking like diffuse red marrow.

Gamesmanship: They can show leukemia in two main ways

(1) Lucent metaphyseal bands in a kid

(2) T1 weighted MRI showing marrow darker than adjacent disks, and muscle.

Remember that Red Marrow is still 40% fat, and should be brighter than muscle on T1.

Most infiltrative conditions affect the marrow diffusely. The exceptions are *multiple myeloma which has a predilection for focal deposits, and Waldenstrom's macroglobulinemia which causes infarcts.*

Chloroma (Granulocytic Sarcoma) - Just say “destructive mass in a bone of a leukemia patient.” It’s some kind of colloid tumor.

Metabolic /Misc

Calcium Hydroxyapatite: Most pathologic calcification in the body is calcium hydroxyapatite, which is also the most abundant form of calcium in bone.

Calcium hydroxyapatite deposition disease = calcific tendinitis.

The calcium is deposited in tendons around the joint. The most common location for hydroxyapatite deposition is the shoulder. Specifically, the **supraspinatus tendon is the most frequent site of calcification**, usually at the insertion near the greater tuberosity. *The longus coli muscle is also a favorite location for multiple choice test writers.* It may be primary (idiopathic) or secondary. Secondary causes worth knowing are: chronic renal disease, collagen-vascular disease, **tumoral calcinosis** and hypervitaminosis D.

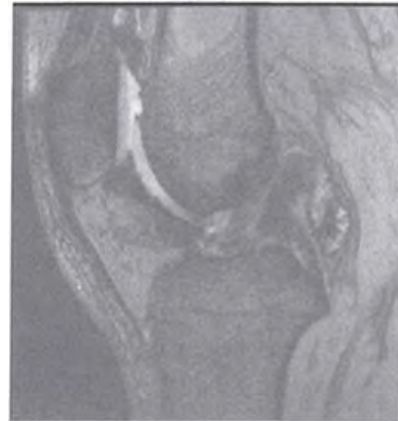
Osteopoikilosis: It’s just a bunch of bone islands. Usually in epiphyses (different from blastic mets or osteosarcoma mets). It can be inherited or sporadic but for the purpose of multiple choice tests *transmission is autosomal dominant.*

Melorheostosis: Characterized by cortical hyperostosis the length of the bone (following a dermatome - but sparing the epiphysis), with the appearance of melted candle wax. The appearance of “endosteal hyperostosis” is often associated. It can be round and resemble osteopoikilosis in the carpal and tarsals. It can actually be symptomatic with pain, swelling, and decreased range of motion. You can even have muscle atrophy and limb length discrepancy. It tends to present in late childhood / early adulthood.

Osteopathia Striata: Linear, parallel, and longitudinal lines in metaphysis of long bones. Doesn't mean shit (usually - but can in some situations cause pain).

Pigmented Villonodular Synovitis (PVNS) : PVNS is an uncommon benign neoplastic process that may involve the synovium of the joint diffusely or focally. It can also affect the tendon sheath.

Intra-Articular Disease : Basically it's **Synovial Proliferation + Hemosiderin Deposition**. The knee is by far the most common joint affected (65-80%). On plain film, features you will probably see are a joint effusion with or without marginal erosions. Osseous erosions with preservation of the joint space, and normal mineralization is typical. It is not possible to distinguish PVNS from *synovial chondromatosis* (see below) on plain film. MRI will be obvious with **blooming on gradient echo**, and this is the most likely way they will show this. Treatment is with complete synovectomy, although recurrence rate is 20-50%.



PVNS - Diffuse Blooming

Trivia: Unusual in kids, but when present is typically polyarticular.

Giant Cell Tumor of the Tendon Sheath (PVNS of the tendon): Typically found in the hand (palmar tendons). Can cause erosions on the underlying bone. Will be soft tissue density, and be T1 and T2 dark (*contrasted to a glomus tumor which is T1 dark, T2 bright, and will enhance uniformly*).

Primary Synovial Chondromatosis: There are both primary and secondary types; secondary being the result of degenerative changes in the joint. The primary type is an extremely high yield topic. It is a metaplastic / true neoplastic process (not inflammatory) that results in the formation of multiple cartilaginous nodules in the synovium of joints, tendon sheaths, and bursa. These nodules will eventually progress to loose bodies. It usually affects one joint, and that one joint is usually the knee (70%). The population are usually a person in their 40s or 50s.

Joint bodies (which are usually multiple and uniform in size) may demonstrate the ring and arc calcification characteristic of chondroid calcification. Treatment involves removal of the loose bodies with or without synovectomy.

PVNS	Synovial Chondromatosis
Benign Neoplasia	Benign Neoplasia
Associated with Hemarthrosis	NOT Associated with Hemarthrosis
Never Calcifies	May Calcify

Secondary Synovial Chondromatosis: A lower yield topic than the primary type. This is secondary to degenerative change, and typically seen in an older patient. There will be extensive degenerative changes, and the fragments are usually fewer and larger when compared to the primary subtype.



Engelmann's Disease: This is also known as progressive diaphyseal dysplasia or PDD. What you see is *fusiform bony enlargement* with sclerosis of the long bones. This is a total zebra that begins in childhood.

Things to know:

- * *It's Bilateral and Symmetric*
- * *It likes the long bones - usually shown in the tibia*
- * *It's hot on bone scan*
- * *It can involve the skull - and can cause optic nerve compression*

Pituitary Gigantism: If they happen to show you x-rays of Andre the Giant, look for **“widening of the joint space in an adult hip”** - can be a classic buzzword. Late in the game the cartilage will actually outgrow its blood supply, and collapse leading to **early onset osteoarthritis**. The formation of endochondral bone at existing chondro-osseous junctions results in widening of osseous structure.

Diabetic Myonecrosis: This is basically infarction of the muscle seen in poorly controlled type 1 diabetics. It **almost always involves the thigh (80%)**, or calf (20%). MRJ will show marked edema with enhancement and irregular regions of muscle necrosis. You **should NOT biopsy this**, it delays recovery time and has a high complication rate.

Soft Tissue Hemangioma: This is a benign vascular tumor, that comes in several varieties (capillary being the most common type). They are more common in women, and *can enlarge during pregnancy*. They can look like a **bunch of phleboliths on plain fdm** (characteristic of the cavernous subtype). On CT you can see **intralesional fat**. On MR they are going to be T1 and T2 bright, again with intralesional fat. They are typically well defined with a lobulated border, and heterogenous features. They enhance avidly and may have blooming on gradient from the phleboliths.

Lipoma Arborescens: This is a zebra that affects the synovial lining of the joints and bursa. The buzzword is “**frond - like**” depositions of fatty tissue. It’s seen in late adulthood (50s-70s), with the most common location being the suprapatellar bursa of the knee. Although it **can develop in a normal knee, it’s often associated with OA, Chronic RA, or prior trauma.** It’s usually unilateral. On MRI it’s going to behave like fat - T1 and T2 bright with response to fat saturation. A sneaky trick is to show this on gradient - and how you pick up the chemical shift artifact at the fat-fluid interface. This could also be shown on ultrasound with a “frond-like hyperechoic mass” and associated joint effusions.

AVN of the Hip: Variety of causes including Perthes in kids, sickle cell, gaucher’s, steroid use etc.... It can also be traumatic with femoral neck fractures (*degree of risk is related to degree of displacement* / disruption of the retinacular vessels). AVN of the hip typically affects the superior articular surface, beginning more anteriorly.

Double Line Sign: Best seen on T2; inner bright line (granulation tissue), with outer dark line (sclerotic bone). Seen in 80% of cases

Rim Sign: Best seen on T2; high T2 signal line sandwiched between two low signal lines. This represents *fluid between sclerotic borders of an osteochondral fragment*, and implies instability. (Stage III).

Crescent Sign: Seen on X-ray (optimally frog leg); Refers to a subchondral lucency seen most frequently anterolateral aspect of the proximal femoral head. It indicates imminent collapse.

Stages of Osteonecrosis:

- o Zero = Normal
- o One = Normal x-ray, edema on MR
- o Two = Mixed Lytic / Sclerotic
- o Three = Crescent Sign, Articular Collapse, Joint Space Preserved
- o Four = Secondary Osteoarthritis

Thalassemia : This is a defect in the hemoglobin chain (can be alpha or beta - major or minor). From the MSK Radiologist prospective we are talking about “hair-on-end” skulls, expansion of the facial bones, “rodent faces” , expanded ribs “jail-bars”. It is frequently associated with extramedullary hematopoiesis.

Thalassemia	Sickle Cell
Will Obliterate Sinuses	Will Not Obliterate Sinuses

Pagets

(High Field Topic)

A relatively common condition that affects 4% of people at 40, and 8% at 80 (*actually 10%, but easier to remember 8%*). M > F. Most people are asymptomatic. The pathophysiology of Pagets is not well understood.

The bones **go through three phases which progress from lytic to mixed to sclerotic** (*the latent inactive phase*). The phrase “**Wide Bones with Thick Trabecula**” make you immediately say Pagets (nothing else really does that).

Lytic	Usually Asymptomatic
Mixed	Elevated Alkaline Phosphate. Fractures
Sclerotic	Elevated Hydroxyproline. More fractures. Sarcomas may develop.

Comes in two flavors: (1) Monostotic and (2) Polyostotic - with the poly subtype being much more common (80-90%).

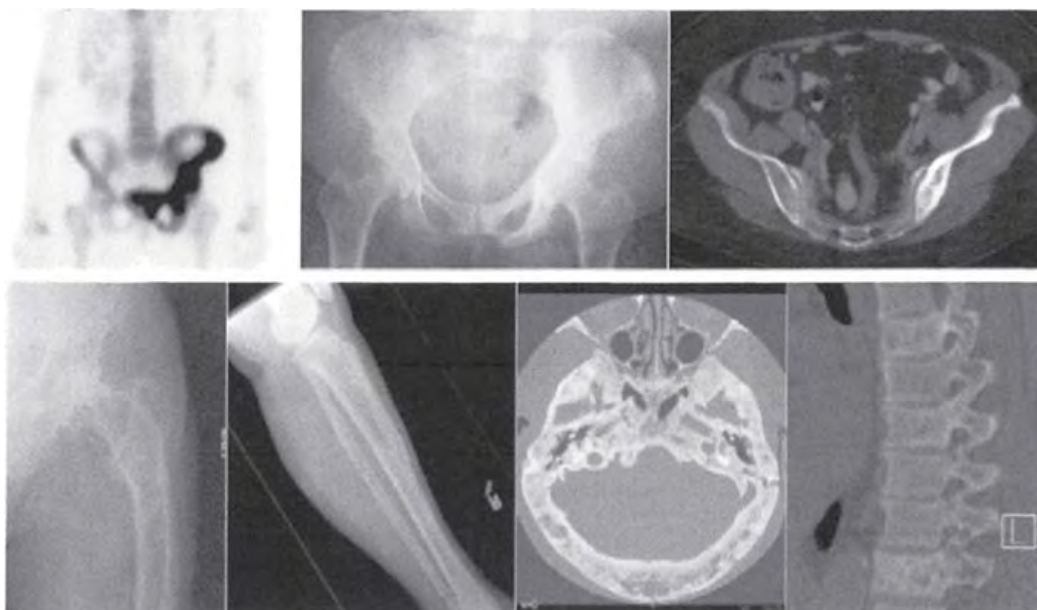
Paget's Buzzwords / Signs:

- * *Blade of Grass Sign:* Lucent leading edge in a long bone
- * *Osteoporosis Circumscripta:* Blade of Grass in the Skull
- * *Picture Frame Vertebra:* Cortex is thickened on all sides (Rugger Jersey is only superior and inferior endplates)
- * *Cotton Wool Bone:* Thick disorganized trabeculae
- * *Banana Fracture:* Insufficiency fracture of a bowed soft bone (femur or tibia).
- * *Tam O 'Shanter Sign:* Thick Skull
- * *Saber Shin:* Bowing of the tibia
- * *Ivory Vertebra:* This is a differential finding, including mets. Pagets tends to be expansile.

Complications: Deafness is the most common complication. Spinal stenosis from cortical thickening is very characteristic. Additional complications, cortical stress fracture, cranial nerves paresis, CHF (high output), secondary hyperparathyroidism (10%), **Secondary development of osteosarcoma (1%) - which is often highly resistant to treatment.** *As a piece of ridiculous trivia - giant cell tumor can arise from pagets.*

Total Trivia: Pagets bone is hypervascular and may be 5 degrees hotter than other bone (get your thermometer ready). Aik Phos will be elevated (up to 20x) in the reparative phase.

Skull	Large Areas of Osteolysis in the Frontal and Occipital Bones “Osteoporosis Circumscripta”, in the lytic phase. The skull will look “cotton wool” in the mixed phase. Favors the outer table.
Spine	Cortical Thickening can cause a “picture frame sign” (same as osteopetrosis). Also can give you an ivory vertebral body.
Pelvis	Most common bone affected. “Always” involves the iliopectineal line on the pelvic brim.
Long Bones	Advancing margin of lucency from one end to the other is the so called “blade of grass” or “flame.” Will often spare the fibula, even in diffuse disease.



Pagets

-- Femur / Tibia, - Expanded Bones, - Coarsened Trabecula, - Ivory Vertebrae

Other Imaging Modalities:

MRI: There are three marrow patterns that closely (but not exactly) follow the phases on x-ray.

Lytic / Early Mixed	Heterogenous T2, T1 is isointense to muscle, with a “speckled appearance”
Late Mixed	Maintained fatty high T1 and T2 signals
Sclerotic	Low signal on T1 and T2

Nuclear Medicine: The primary utility of a bone scan is in defining the extent of disease and to help assess response to treatment. The characteristic look for Pagets is “Whole Bone Involvement.” For example, the **entire vertebral body including the posterior elements**, or the entire pelvis. The classic teaching is that Pagets is hot on all three phases (although often decreased or normal in the sclerotic phase).

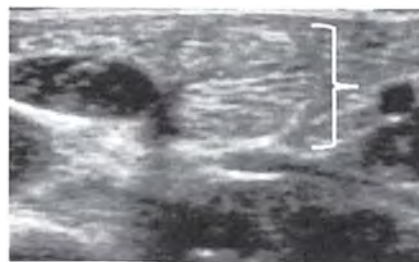
Tendon Ultrasound:

It’s absolutely incredible that I even need to go over this, but dinosaur radiologist’s love this stuff.

Anisotropy: The most common and most problematic issues with ultrasounding tendons is this thing called “anisotropy.” The tendon is normally hyperechoic, but if you look at it when it’s NOT perpendicular to the sound beam it can look hypoechoic (injured?).

It’s the biggest pain in the ass:

- * Supraspinatus tendon - as it curves along the contours of the humeral head
- Long Head of the Biceps - In the bicipital groove



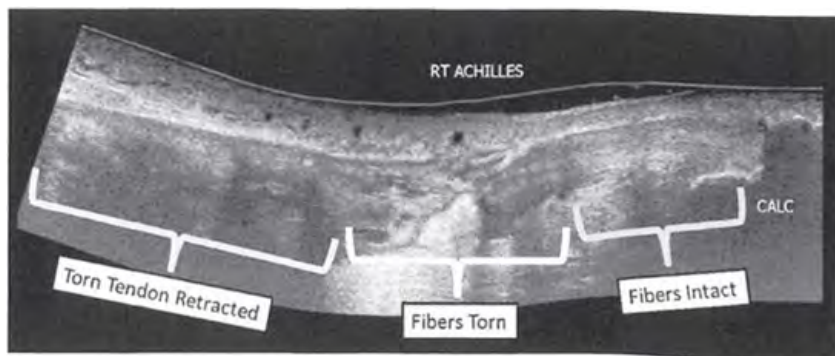
Normal
Appearing
Hyperechoic
Tendon



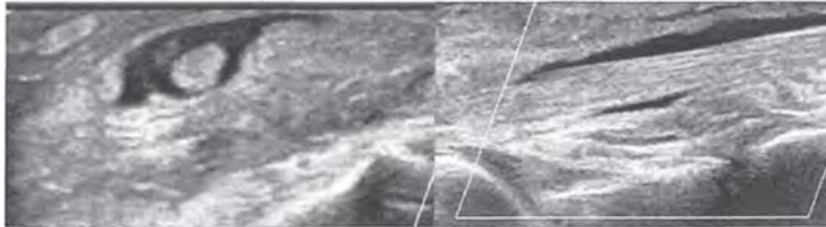
Exact same
tendon - now
appearing
Hypoechoic -
when scanned
non-parallel

Anisotropy

Tears: The tendon is usually hyperechoic. Focal hypoechoic areas are tears. It can be really tricky to tell if it’s partial or complete (that’s what MRI is for).



Tenosynovitis: As discussed above there are a variety of causes. If they show it on ultrasound you are looking for increased fluid within the tendon sheath. You could also see associated peritendinous subcutaneous hyperemia on Doppler.

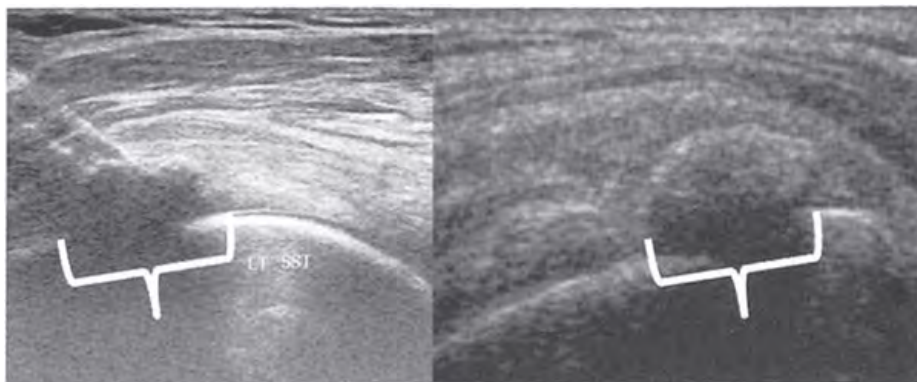


Tenosynovitis - Increased fluid in the tendon sheath

Plantar Fasciitis: This is another pathology that lends itself to a “what is it ?” type of ultrasound question. Hopefully, they at least tell you this is the foot (they could label the calcaneus). The finding will be thickening of the plantar fascia (greater than 4mm), with loss of the normal fibrillar pattern. If you see calipers on the plantar fascia - this is going to be the answer.



Calcific Tendonitis: As described above, this is very common and related to hydroxyapatite. The most common site is the supraspinatus tendon, near its insertion. It will shadow just like a stone in the GB.

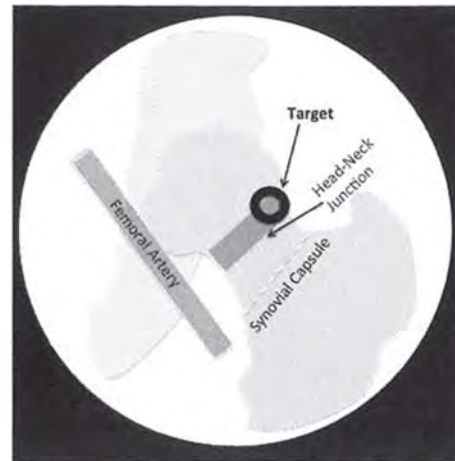


Calcific Tendonitis-Shadowing calcification in the classic location (supraspinatus)

Basic Procedural Trivia - The Arthrogram

An important point to remember is that the target is not actually the joint. The target is the capsule. In other words, you just need the needle to touch a bone within the capsule. The trick is to do this without causing contamination or damaging an adjacent structure (like an artery). *General Tip* - Avoid putting air in the joint, this will cause susceptibility artifact.

Hip: The general steps are as follows: (1) Mark the femoral artery. (2) Internally rotate the hip (slightly) to localize the femoral head-neck junction (your target). (3) Clean and numb the skin. (4) Advance a 20-22 gauge spinal needle into the joint - straight down on the superior head neck junction. (5) Inject a small amount of contrast to confirm position. Contrast should flow away from the tip. If the contrast just stays there it's not in a space. (6) Put the rest of the contrast in.



Trivia: The capsule is widest at the head-neck junction.

Trivia: The cocktail injected is around 14cc total (4cc Lidocaine, 10cc Visipaque, and only about 0.1 cc Gd).

Shoulder: The general steps are as follows: (1) Supinate the hand (externally rotate the shoulder) (2) Clean and numb the skin. (3) Advance a 20-22 gauge spinal needle into the joint - straight down on the junction between the middle and inferior thirds of the humeral head - 2mm inside the cortex. (4) Once you strike bone, pull back 1mm and turn the bevel towards the humeral head - this should drop into the joint (5) Inject a small amount of contrast to confirm position. Contrast should flow away from the tip. If the contrast just stays there it's not in a space. (6) Put the rest of the contrast in.



Trivia: The cocktail injected is around 12cc total (4cc Lidocaine, 8cc Visipaque, and only about 0.1 cc Gd).

13 Nuclear Medicine

Prometheus Lionhart, M.D.



Nuclear medicine is probably the most challenging section on the Exam (maybe secondary to physics). The reason it's so difficult is that there is a seemingly unending amount of trivia, very little of which is necessary to understanding or interpreting the exam, but nonetheless lends itself easily to multiple choice test writing.

High Yield Topics:

- *“ What Scan is It? ”*
- *Thyroid Treatment*
- *RBC Labeling*
- *False Positive / Negative on PET*
- *Cardiac Pharmacology*
- *Therapy - especially bone met therapy*

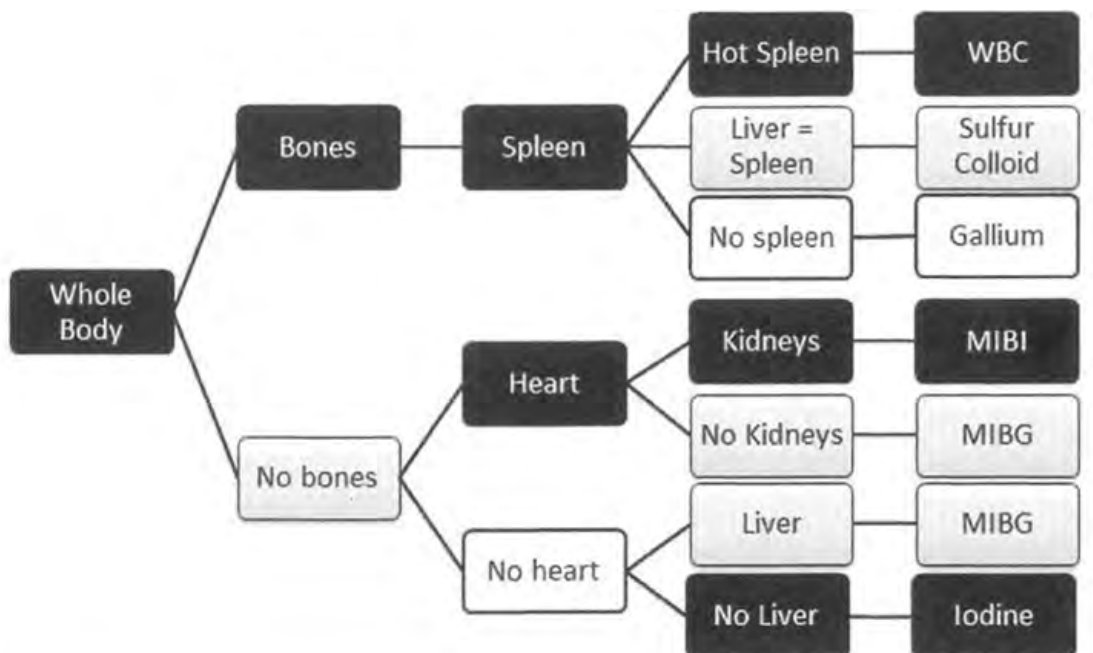
What Scan Is That?

Ok, here is the scenario that I want you to be prepared for:

The plane has crashed. All the nuclear techs are dead. Prior to the plane crashing, they completed several studies, but forgot to label them or give indications. The bean counter (non-MD) who is running the hospital is breathing down your neck to read these studies now, because the metrics he set up are gonna look bad at the next QA/QC meeting. So now you have to interpret nuclear studies, and you don't know why they did it or even what tracer was given.

Fortunately, you trained for this as part of your preparation for the CORE Exam.

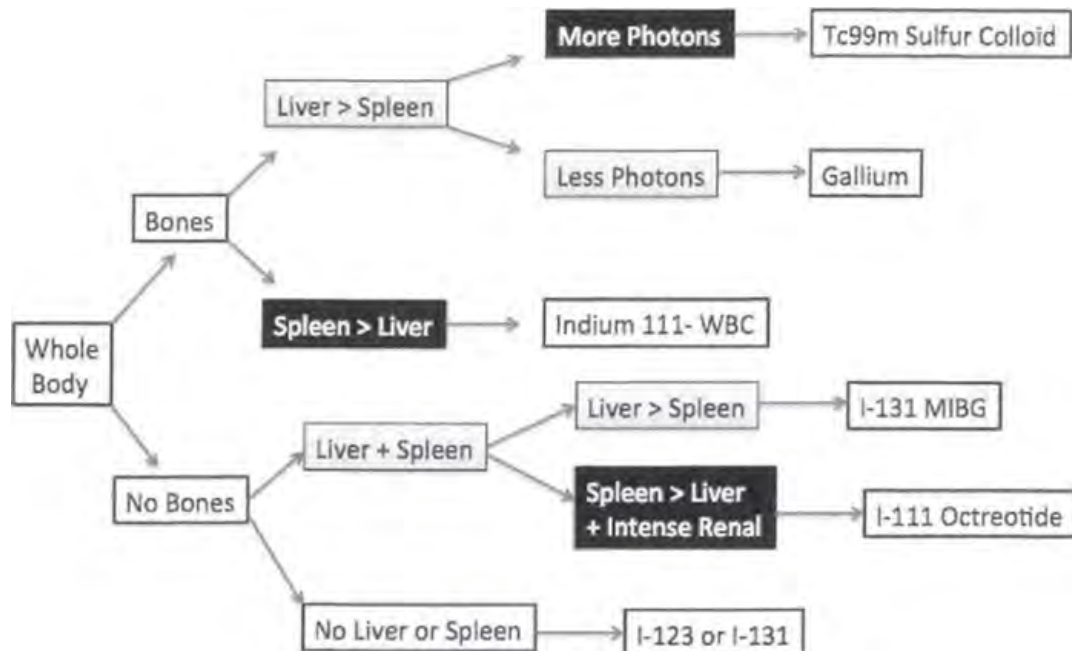
Seriously, this is famously one of the most common ways nuclear medicine is tested. It was like that on the old oral boards, and it is still like that now (same knuckle heads writing the questions). It's such a ridiculous thing to ask. My primary advice: (1) *Don't Fight It...* Embrace the Ridiculous Nature of the Test. (2) Get a private practice job and run your rival academic center out of business.



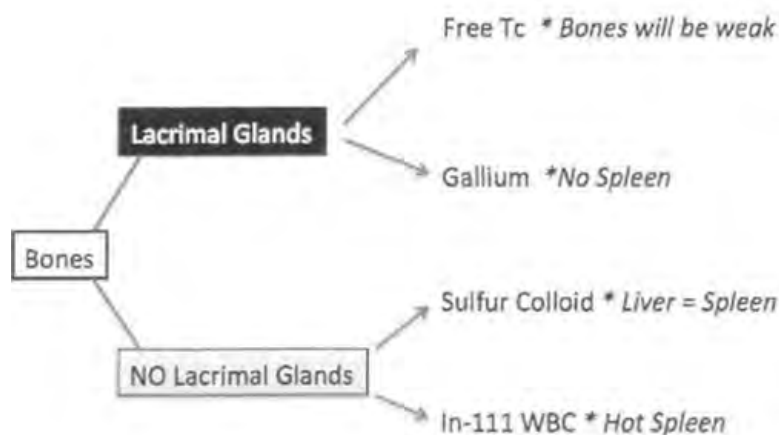
Octreotide = Higher Count Study - VERY hot Spleen and Kidneys

** Note the MIBG is under both heart and no heart - this is because it's variable. MIBG with 1-123 is more likely to have heart than 1-131.*

This is an alternative pathway that some people prefer. This one focuses more on photon output (how light or dark stuff is), and liver and spleen. It removes the confusion of heart “maybe” for MIBG.



Another alternative way to work the bones pathway is to ask Lacrimal Glands? Gallium will have them, WBC scans and Sulfur Colloid will NOT. The trick on Lacrimal Glands is free Tc (but bones will be real weak on that one). MIBG can have lacrimal activity , but again no bones.



HOT Spleen: You should think Octreotide, and WBC Scans. Sulfur Colloid will have tracer in the spleen, but not as much as the liver.

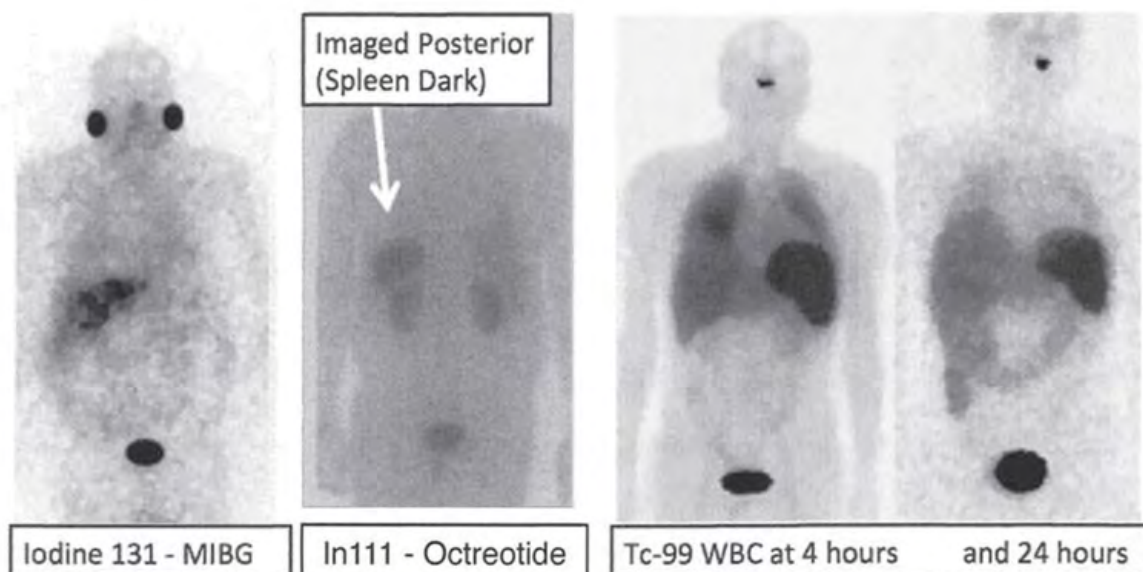
MIBG: It has variable cardiac uptake, so it finds itself on multiple branch points. Cardiac activity is more often seen on an 1-123 MIBG scan (as opposed to 1-131 MIBG). Another thing that helps me remember this stuff: when you do a MIBG you are often looking for neuroblastoma. If the kidney was also hot it would be hard to tell a mass near the kidney from the kidney - so part of the reason the study works is that the kidney does NOT take up MIBG.

Octreotide: This is a high count study, images should be cleaner. You can go down the “no bones” pathway. But the trigger should be **no bones + liver + dark spleen + dark kidneys**.

Tc WBC: The trick here can be imaging at 4 hours vs imaging at 24 hours. At 4 hours you can see lung uptake. At 24 hours the lungs are clearing up, but you start to get some bowel uptake. Just like an In-WBC the spleen is still darker than the Liver.

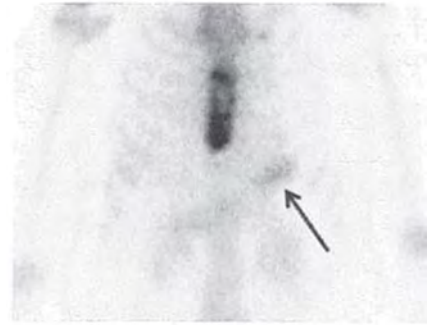
In WBC vs Tc WBC: Both will have hot spleens. Additionally, Tc is a higher count study and will typically look cleaner.

Tc WBC	In WBC
Renal	NO Renal
GI	NO GI



Skeletal Nuclear Medicine

The workhorse of skeletal imaging is methylene diphosphonate (MDP) tagged with Tc-99m. This is prepared from a kit which has MDP and stannous ion. You add free pertechnetate and the stannous ion reduces it so it will bind to the MDP. If you don't have enough stannous ion (or you get air into the vial or syringe - that can cause oxidation) you might get free Tc (salivary gland, thyroid, stomach uptake). After you inject the tracer (15-25mCi) you wait 2-4 hours to let the tracer clear from the soft tissues (so you can see the bones).



Free Tc: - Gastric Uptake on bone scan
* incidental note of sternal met from breast ca

A brief discussion of F-18 vs Tc-MDP

The take home point is F-18 PET is way way way better than Tc-MDP. The image quality and sensitivity of F18 is multiple orders of magnitude better than Tc-MDP. It also has a shorter examination time. So, why do you never see F-18? Because it costs more, and insurances won't pay etc... Politics and Finance are the reasons. It's common gamesmanship to ask you to tell the difference between the two (discussed below). Another thing they can ask is what organ gets the highest dose? The **organ receiving the highest dose is Bone with MDP, and Bladder with F-18.**

Scan on Scan on Scan

This is a common trick, popular in case books & case conferences - asking you to distinguish F-18 bone scan vs Tc-MDP bone scan vs PET-FDG with marrow stimulation.

This is how you do it:

- * **Tc-MDP** will have bone and kidney uptake. It will be a blurry fuzzy piece of crap.
- * **F-18** will be beautiful, super high resolution, and look like a MIP PET.
- * **FDG -PET with bone stimulation** - will look similar to the F-18, but will have brain uptake.

What Factors will affect tracer uptake?

- * OsteoBLASTIC activity (why pure lytic lesions can be cold)
- * Blood Flow

Where is tracer uptake NORMAL ?

- * Bone (duh)
- * Kidney (not seen = Super Scan)
- * Bladder
- * Breasts (especially in young women)
- * Soft tissues - low levels
- * Epiphyses in kids

Let's Talk About Abnormal Distribution:

Increased focal uptake is very nonspecific, and basically is just showing you bone turn over. So a metastatic deposit can do that (and this is the classic indication). But, you can also see it with arthritis (classically shoulder) and healing fractures (most commonly shown with segmental ribs).

Some Sneaky Situations:

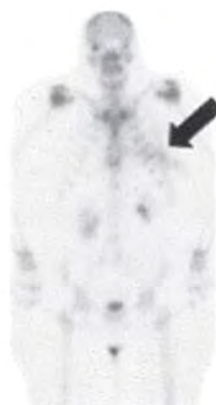
- * *Skull Sutures:* It's normal to see some persistent visualization of the skull sutures, **BUT** when this is **marked you may be thinking about renal osteodystrophy.**
- * *Breast Uptake:* Some mild diffuse breast uptake is normal (especially in younger women), **BUT focal uptake can be cancer.**
- * *Renal CORTEX activity:* You are suppose to have renal activity (not seeing kidneys can make you think super scan), **BUT** when the **renal cortex is hotter than the adjacent lumbar spine** you should **think about hemochromatosis.**
- * *Diffuse Renal Uptake:* This often occurs in the setting of chemotherapy (especially if the study is looking for bone mets). This also can be seen with urinary obstruction.



Multiple Continuous Lesions
(Rib Fractures)



Asymmetric Breast Tissue Uptake
(Primary Breast CA)



Diffuse Renal Uptake
(Chemotherapy)

Liver Uptake: This can be several things, but the main ones to think about are (1) **Too Much Al+3** contamination in the Tc, (2) Cancer - either primary **hepatoma** or mets, (3) **Amyloidosis**, (4) **Liver Necrosis**

- *Spleen Uptake:* This is a common trick to show an **auto-infarcted spleen** - common in **sickle cell patients**. These same patient's are going to have scattered hot and cold areas from multiple bone infarcts.

- * *The Single Lesion:* When you see a single hot lesion, the false positive rate for attributing the finding to a met is high. Only about 15% to 20% of patients with proven mets have a single lesion (most commonly in the spine). In other words **80% of the time it's benign. A classic exception is a single sternal lesion in a patient with breast cancer**. This is due to breast CA 80% of the time.



"Honda Sign"
Sacral Insufficiency Fracture

- * *Sacral Insufficiency fracture:* This is a hot geographic area, confined to the sacrum, often with a characteristic butterfly or **"H" shaped (Honda sign)**. Osteoporosis is the most common cause, but it can also occur in a patient who has had radiation.
- * *Diffusely Decreased skeletal uptake:* This can be seen with (1) free Tc, or (2) Bisphosphonate therapy.
- * *Fractures in the Elderly (including elder abuse):* In older populations, bone scans may be negative for several days. A bone scan obtained at **1 week** will exclude a fracture.

Flair Phenomenon: This is a sneaky situation shown on bone scan, where a good response to therapy will mimic a bad response. What happens is you have increased radiotracer uptake (both in number and size of lesions) seen 2 weeks to 3 months after treatment.

So how can you tell it's flair and not actually cancer getting worse?

- * On plain film lesions should get more sclerotic
- * After 3 months they should improve.

Specific Cancer - Specific Trivia

- * Prostate Cancer Loves Bone Mets (85% of dying patients have it)
- * Prostate Cancer bone mets are uncommon with a PSA less than 10 mg/ml
- * Lung Cancer bone mets tend to be in the appendicular skeleton
- * Lung Cancer can have hypertrophic osteoarthropathy (10%)
- * Breast Cancer bone mets are most common to the spine, but the solitary sternal lesion is more specific
- * Neuroblastoma frequently mets to the bones (metaphysis of long bones)
- * 1-123 and 131 MIBG is superior for detection of neuroblastoma bone mets

Cold Lesions

- * Radiation Therapy (usually segmental)
- * Early Osteonecrosis
- * Infarction (*very early or late*)
- * Anaplastic Tumor (Renal, Thyroid, Neuroblastoma, Myeloma)
- * Artifact from prosthesis
- * Hemangioma

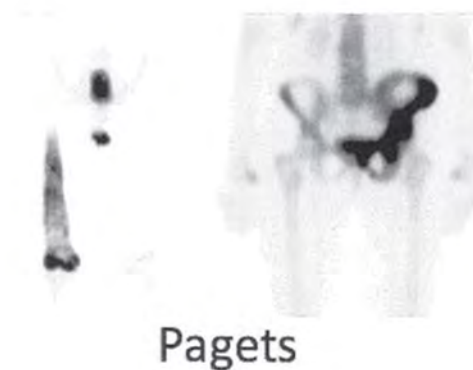
Bone Scan vs Skeletal Survey (Trivia)

- * Bone Scan is way better (more sensitive) than skeletal survey when dealing with blastic mets
- * Skeletal Survey is superior (more sensitive) for lytic mets
 - o “Skeletal survey is the preferred evaluation for osseous involvement in multiple myeloma”

Hypertrophic Osteoarthropathy: This is a **“Tramline”** along the periosteum of long bones, which is associated with conditions of chronic hypoxia (CF, Cyanotic Heart Disease, Mesothelioma, Pneumoconiosis). However, when you see this - you need to think **lung cancer**. Apparently it’s actually seen in 10% of patients with lung cancer.



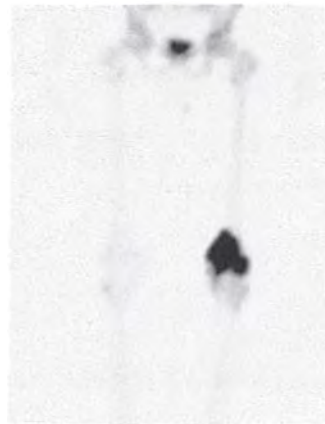
Pagets: Seen primarily in older patients (8% at 80), it’s classically shown five ways: (1) Super Hot Enlarged Femur (2) Super Hot Enlarged Pelvis, (3) Super hot skull, (4) Expanded hot “entire” vertebral body (5) metabolic superscan - from widespread Pagets. As a point of gamesmanship if they show you a metabolic superscan the answer is probably hyper PTH.



Pagets Spine - Classically involves BOTH the vertebral body and posterior elements.

Primary Bone Tumors: Both Osteosarcoma and Ewings will be hot. Primary utility of the bone scan is to see extent of disease. With regard to benign bone tumors the only ones worth knowing are the HOT ones and the COLD ones. Osteoid Osteoma is worth knowing a few extra things about (because they lend themselves easily to multiple choice questions).

Osteoid Osteoma: The lesion will be focal and three phase hot. A central hot nidus is often seen (**double density** or **hotter spot within hot area**). A normal bone scan excludes this entity.



Osteosarcoma



Osteoid Osteoma
-Double Density

Fibrous Dysplasia: *Be aware that in case books / case conference this is sometimes shown as a super hot mandible. Could also be shown as a leg, that looks similar to pagets. Benign Lesions on Bone Scan:*

- | | |
|---|---|
| <ul style="list-style-type: none"> • "HOT" (<i>intense</i>) <ul style="list-style-type: none"> • Fibrous Dysplasia • Giant cell tumor • Aneurysmal bone cyst • Osteoblastoma • Osteoid osteoma | <ul style="list-style-type: none"> • "COLD" <ul style="list-style-type: none"> • Bone cyst without fracture • VARIABLE <ul style="list-style-type: none"> Hemangioma Multiple hereditary exostosis |
|---|---|

Heterotopic Ossification: The main reason you image this is to see if it's "mature" or not. Serial exams are used to evaluate if the process is active or not. If it's still active it has a higher rate of recurrence after it's resected. The idea is you can follow it with imaging until it's mature (cold), then you can hack it out (if someone bothers to do that).

Avascular Necrosis: AVN can occur from a variety of causes (EtOH, Steroids, Trauma, Sickle Cell, Gauchers). The trick on bone scan is the timing. **Early and late AVN is cold. Middle (repairing) will be hot.**

Super Scans

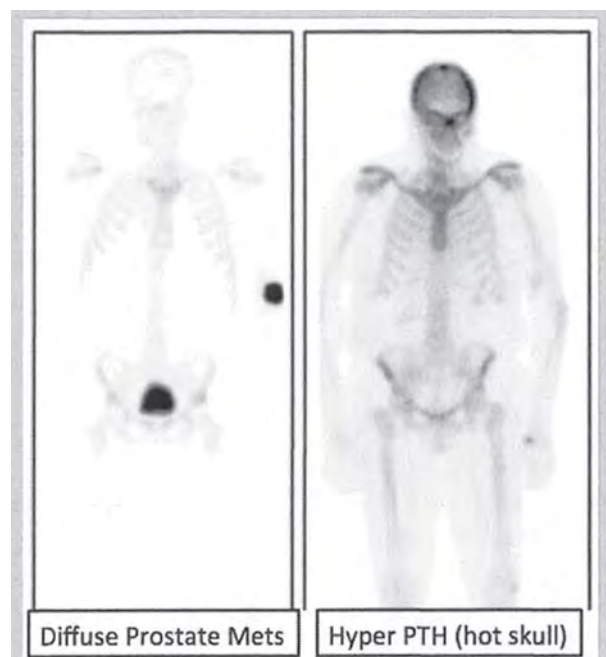
This is a common trick, where the scan shows no abnormal focal uptake, but you can't see the kidneys. The trick is that everything is hot.

This occurs in two flavors:

- * Diffuse Mets: Diffuse skeletal metastatic activity (breast and prostate are the common culprits).
- * Metabolic: From metabolic bone pathology; including *hyper parathyroid*, renal osteodystrophy, pagets, or severe thyrotoxicosis.

How can you tell them apart?

- The Skull will be asymmetrically hot on the metabolic super scan.



Super Scans

Don't get it twisted - A common sneaky move is to show you a bone scan, with no renals. But it's because there is a horseshoe kidney in the pelvis. Could be phrased as a next step question, with the answer being look at prior CT to confirm normal anatomy.

BAD BONES!

Any bone uptake on MIBI, 1-131, or Octreotide is abnormal, and concerning for skeletal mets.

Equivocal Lesion Next Step?

If a bone scan "equivocal lesion" is found the next step is a plain film. If the plain film shows no corresponding lesion this is MORE suspicious for mets. Next step at that point would be a MRI.

The Three Phase Scan:

Bone scans can be done in a single delayed phase, or in 3 phases (flow, pool, and delayed). A lot of things can be “3 phase hot”, including osteomyelitis, fracture, tumor, osteoid osteoma, charcot joint, and even reflex sympathetic dystrophy.

Cellulitis vs Osteomyelitis: The benefit of using 3 phases is to distinguish between cellulitis (which will be hot on flow and pool, but not delays), and osteomyelitis (which is 3 phase hot). In children a whole body bone scan is often performed to evaluate extent. Additionally, because of subperiosteal pus/edema you can actually have decreased vascularity to the infected area (cold on initial phases) but clearly hot on delayed phases.

In the spine, gallium (combined with bone scan) or MRI are the preferred imaging modalities.

Response: You can also use a bone scan to evaluate response to treatment. Blood flow and blood pool tend to stay abnormal for about 2 months, with delayed activity persisting for up to 2 years. This is especially true when dealing with load bearing bones. Gallium⁶⁷ and Indium¹¹¹ WBC are superior for monitoring response to therapy.

Reflex Sympathetic Dystrophy (RSD): Sometimes called “complex regional pain syndrome,” it can be seen after a stroke, trauma, or acute illness. The classic description is **increased uptake on flow and blood pool, with periarticular uptake on delayed phase.** The uptake often involves the entire extremity. About one third of adult patients with documented RSD do not show increased perfusion and uptake (*which probably means they are faking it, and need a rheumatology consult for fibromyalgia*). In children, sometimes you actually see decreased uptake.

Sulfur Colloid Bone Scan & WBC Imaging:

Tc can be tagged to sulfur colloid with the idea of getting a normal localization to the bone marrow. You can actually perform Tc sulfur colloid studies to map the bone marrow in patients with sickle cell (with the idea to demonstrate marrow expansion and bone infarct). However, the major utility is to use it in combination with tagged WBC or Gallium.

Both Tc Sulfur Colloid and WBCs will accumulate in normal bone marrow, in a spatially congruent way (they overlap). The principal is that infected bone marrow will become photopenic on Tc-Sulfur Colloid. Now, this takes about a week after the onset of infection, so you have to be careful in the acute setting. WBC on the other hand will obviously still accumulate in an area of infection. *Combined Tc-Sulfur Colloid and WBC study is positive for infection if there is activity on WBC image, without corresponding Tc activity on the bone marrow image.* When imaging the spine, WBC frequently fails to migrate showing a photopenic area. This is why **gallium is preferred for osteomyelitis of the spine.**

Prosthesis Evaluation: Differentiating infection from aseptic loosening is challenging and the most common reason a nuclear medicine doctor would get involved in the situation. Bone scan findings of periprosthetic activity is very nonspecific, because you can see increased tracer activity in a hip up to 1 year after placement (even longer in cementless arthroplasty). Typically, there would be diffusely increased activity on imaging with Tc-MDP in the case of infection (**more focal along the stem and lesser trochanter with loosening**) - but this isn't specific either. Combined Tc-Sulfur Colloid and WBC imaging is needed to tell the difference.

Helpful when negative - A negative bone scan excludes loosening or infection.

Neuropathic Foot: Most commonly seen in the tarsal and tarsal-metatarsal joints (60%), in diabetics. When the question is infection (which diabetics also get), it's difficult to distinguish arthritis changes vs infection with Tc-MDP. Again, combined marrow + WBC study is the way to go.

An additional pearl that could make a good "next step" question is the need for a fourth phase in diabetic feet. As these patient's tend to have reduced peripheral blood flow, the addition of a 4th phase at 24 hours may help you distinguish between bone and delayed soft tissue clearance.

Instead of In-WBC, What about Tc99 HMPAO WBC ?

When would you consider Tc99 HMPAO instead of In-WBC for infection ? Two main reasons

- (1) Kids - Tc99 will have a *lower absorbed dose & shorter imaging time*, and
- (2) Small Parts - Tc99 does better in hands and feet

Why not use Tc99 HMPAO all the time ? The downsides to Tc99 HMPAO are

- (1) It has a shorter half life -6hrs- which limits delayed imaging, and
- (2) It has normal GI and gallbladder activity which would obscure injection in those areas.

Pulmonary Nuclear Medicine

If 1940 calls and wants to rule out a PE you'll want to get the angiography room ready. In 2014, textbooks and papers still frequently lead with the following statement "*Pulmonary angiography is the definitive diagnostic modality and reference standard in the diagnosis of acute PE.* " In reality, pulmonary angiography is almost never done, and CTPA is the new diagnostic test of choice. V/Q scan is usually only done if the patient is allergic to contrast or has a very low GFR. The primary reason V/Q isn't done, is that it's often intermediate probability, and the running joke is that if you don't know how to read one, just say it's intermediate and you'll probably be right.

The idea behind the test is that you give two tracers: one for ventilation and one for perfusion. If you have areas of ventilated lung that are not being perfused that may be due to PE. Normally Ventilation and Perfusion are matched, with a normal gradient (less perfusion to the apex - when standing).

Tracers:

Perfusion: For perfusion Tc-99m macroaggregated albumin (**Tc-99m MAA**) is the most common tracer used. MAA is prepared by heat denaturation of human serum albumin, with the size of the particles commercially controlled. You give it IV and the tracer should stay in the pulmonary circulation (vein-> SVC-> right heart -> pulmonary artery -> lung *STOP). The tracer should light up the entire lung. A normal perfusion study excludes PE. Areas of perfusion abnormality can be from PE or other things (more on this later). The biologic half life is around 4 hours (they eventually fall apart, becoming small enough to enter the systemic circulation to eventually be eaten by the reticuloendothelial system).

Ventilation: There are two ways to do the ventilation; you can use a radioactive gas (Xenon-133) or a radioactive aerosol (Tc-99m DTPA).

- * **Xenon 133:** Also the physical half-life is 5.3 days, the **biologic half-life is 30 seconds** (you breath it out). Because it has low energy (80 keV) it **is essential to do this part of the test first** (*more on this in the physics chapter*). Additionally, because the biologic half life is so short you only can do one view (usually posterior), with a single detector (dual detector can do anterior and posterior). There are 3 phases to the study: (1) wash in (single max inspiration and breath hold), (2) equilibrium (breathing room air and xenon mix), and (3) wash out (breathing normal air)
- * **Tc-99m DTPA:** This one requires patient cooperation: because they have to breath through a mouth guard with a nose clamp for several minutes. It is also essential to do this part of the test first.

5 Classic Trivia Questions about Tc99m MAA:

- (1) *They show tracer in the brain:* This a classic way of showing you a shunt (it got into the systemic circulation somehow, maybe an ASD, VSD, or Pulmonary AVM).
- (2) *How big are the particles?* A capillary is about 10 micrometers. You need your particles to stay in the lung, so they can't be smaller than that. You don't want them to be so big they block arterioles (150 micrometers). So the answer is 10-100 micrometers.
- (3) *When do you reduce the particle amount?* A few situations. You don't want to block more than about 0.1% of the capillaries, so anyone who has fewer capillaries (children, people with one lung). Also you don't want to block capillaries in the brain, so anyone with a right to left shunt. Lastly anyone with pulmonary hypertension.
- (4) *Is reduced particle the same as reduced dose?* Nope. The normal dose of Tc can be added to fewer particles.
- (5) *They show you multiple focal scattered hot spots:* This is the classic way of showing "clumped MAA", which happens if the tech draws blood into the syringe prior to injection.

Classic Trivia Questions for Xenon 133:

- (1) *They show you persistent pulmonary activity during washout:* This indicates Air Trapping (COPD)
- (2) *They show you accumulation of tracer over the RUQ:* This is fatty infiltration of the liver (xenon is fat soluble).

Classic Trivia Questions for Tc-99m DTPA

Xenon	TC-99m DTPA
Quick Wash Out only one or two views	Slower Wash Out - multiple projections
Activity homogenous in the lungs	"Clumping" common in the mouth, central airways, and stomach (from swallowing).

Interpretation:

The money for PE is normal ventilation with multiple areas of abnormal perfusion. A normal perfusion essentially excludes PE. You always need a CXR (typically done within 24 hours of the VQ). Pneumonia or atelectasis will obviously not ventilate well, but can also shunt blood away from that “un-used” part of the lung. The pearl is the pulmonary infarct will result in an opacity on CXR, a ventilation defect, and a perfusion defect and therefore this combination is referred to as a “Triple Match.” When in the lower lung fields a “Triple Match” is considered intermediate. The perfusion is also better in the bases (should never get lighter going down on the perfusion).

Reverse Mismatch: Normal Perfusion with Abnormal Ventilation. This is most commonly due to atelectasis. It can also be seen with pleural effusion, pneumonia, cardiomegaly, and partial bronchial obstruction.

Stripe Sign: This describes a perfusion abnormality with a zone of normally preserved peripheral lung. Because PE is usually peripherally based, this finding makes PE unlikely and considered very low probability.



Fissure Sign: This describes curvilinear perfusion defects, which correspond with pleural fluid in the fissures.

Solitary Lobar or Solitary Whole Lung Perfusion Defects: Think hilar mass, hypoplastic pulmonary artery, or mediastinal fibrosis. It's uncommon for a PE to present like this (they are usually multiple and bilateral).

Testable Trivia: Most common cause of unilateral whole lung perfusion defect with normal ventilation = lung cancer.

Pleural Effusions: Pleural effusions can be tricky in that they produce reverse mismatch defect or triple match defects.

- * Small pleural effusions causing matched defects = intermediate probability
- * Large pleural effusions = low probability
- * Triple Matched Defects in the Lower Lobe (caused by any size effusion) = intermediate

o *Triple Matched Defects in the Middle and Upper lobe = Low probability

Follow up: After a high probability scan, follow up in 3 months is recommended to establish a new baseline. The rationale is that perfusion defects that persist at 3 months are usually permanent.

Quantitative Perfusion:

You can do quantitative studies typically to evaluate prior to lung resection, or prior to transplant. You want to make sure that one lung can hold its own, if you are going to take the other one out.

Testable Trivia: Quantification is NOT possible if you use Tc-99m DTPA aerosol. You can do it with a combined Xe + Tc MAA because the Xe will not interfere with the Tc.

Pulmonary Infections

Using Gallium:

Gallium ⁶⁷ Scan

The body handles Ga^{+3} the same way it would Fe^{+3} - which as you may remember from step 1 gets bound (*via lactoferrin*) and concentrated in areas of inflammation, infection, and rapid cell division. Therefore it's a very non-specific way to look for infection or tumor. Back in the stone ages this was the gold-standard for cancer staging (now we use FDG-PET). I should point out that Gallium can also bind to neutrophil membranes even after the cells are dead, which gives it some advantages over Indium WBC - especially in the setting of chronic infection.

Gallium is produced in a cyclotron via the bombardment of Zn^{68} , at which point it's complexed with citric acid to make Gallium Citrate. The half life is around 3 days (78hours). It decays via electron capture, emitting gamma rays at 4 photopeaks:

93 keV - 40%
184 keV - 20%
300 keV - 17%
393 keV - 5%

Images are not typically done sooner than 24 hours - because background is too high. *The target organ is the colon.*



Normal localization: Liver (*which is the highest uptake*), bone marrow ("*Poor Man's bone scan*"), spleen, salivary glands, lacrimal glands, breasts (especially if lactating, or pregnancy). Kidneys and bladder can be seen in the first 24 (faintly up to 72 hours). Faint uptake in the lungs can be seen in < 24 hours. After 24 hours you will see some bowel. In children the growth plates and thymus.

"Poor Man's Bone Scan" - Uptake is in both cortex (like regular bone scan) and marrow. Degenerative change, fractures, growth plates, all are hot - just like bone scan.

Renal Uptake - Normal at 24 hours. Activity after 48 hours should be considered suspicious.

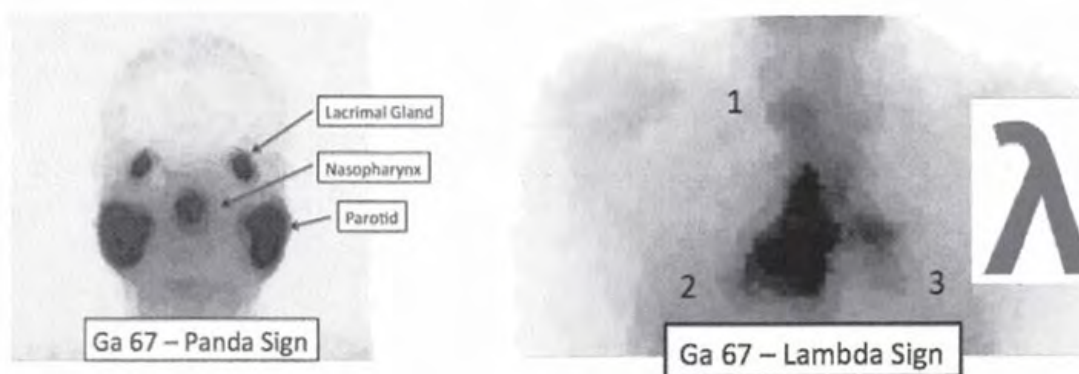
Uptake is nonspecific and can be seen with a variety of things including infection, but also CHF, atelectasis, and ARDS.

Sarcoidosis:

The utility of Gallium in Sarcoidosis patients is to help look for active disease. Increased uptake in the lungs is 90% sensitive for active disease (scans are negative in inactive disease). Additionally, Gallium can be used to help guide biopsy and lavage - if looking to prove the diagnosis. The degree of uptake is graded relative to surrounding tissue (greater than lung is positive, less than soft tissue is negative).

Classic Signs:

- * *Lambda Sign* - The nuke equivalent to the “1-2-3 Sign” on Chest x-ray. You have increased uptake in the bilateral hila, and right paratracheal lymph node.
- * *Panda Sign* - Prominent uptake in the nasopharyngeal region, parotid salivary gland, and lacrimal glands. This can also be seen in Sjogrens and Treated Lymphoma.

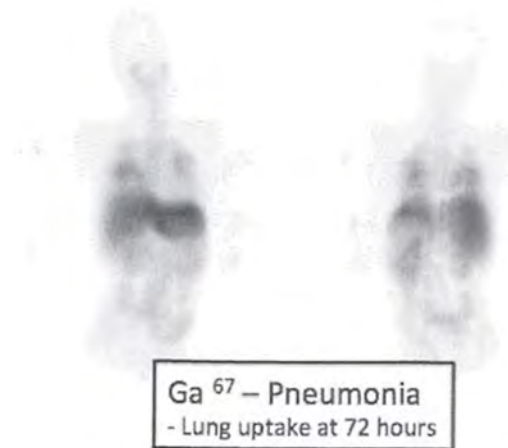


Other Noninfectious Things

- * Gallium can be used to show early drug reaction from chemotherapy (Bleomycin) or other drugs (Amiodarone).
- * Gallium is elevated in IPF (idiopathic pulmonary fibrosis) and can be used to monitor response to therapy.

Immunosuppressed Patients

- * *PCP* - Gallium Hot, —
Characteristic Gallium Pattern is
Diffuse Bilateral Pulmonary
Uptake
- * *Kaposi Sarcoma* - Gallium
Negative, Thallium Positive
- * *Bacterial Pneumonia* - Intense
lobar configuration without
parotid or nodal uptake



Misc Infections That Gallium Can Pick Up:

- *Abdominal and Pelvic Infections* - In-111 WBC is superior to Gallium (Gallium has some normal GI uptake).
- *Malignant Otitis Media* - Will be both Gallium and Bone Scan (Temporal Bone) Hot.
- *Spinal Osteomyelitis* - Gallium is superior to Indium WBC for spinal infections.

Thyroid Imaging / Treatment

The thyroid likes to drink Iodine (it's sort of its job). Imaging takes advantage of this with Iodine analogs. The distinction between "trapping" and "organification" is a common question.

- * *"Trapping"* - Analog is transported into gland. ^{123}I , ^{131}I , and $^{99\text{m}}\text{Tc}$ all do this.
- * *"Organification"* - Analog is oxidized by thyroid peroxidase and bound to tyrosyl moiety. ^{123}I and ^{131}I do this. **$^{99\text{m}}\text{Tc}$ does NOT do this.** Instead $^{99\text{m}}\text{Tc}$ slowly washes out of the gland.

Tracer options / pros and cons.

I-131: The major advantage here is that it's cheap as dirt. The disadvantage is that it has a long half life (8 days), and that it's a high energy (364 keV) beta emitter. The high energy makes a crappy image with a *Vi* inch crystal. It's ideal for therapy, not for routine imaging. It's contraindicated in kids and pregnant women.

I-123: This guy has a shorter half life (13 hours) and ideal energy (159 keV). It decays via electron capture and all around makes a prettier image. The problem is that it costs more.

Tc-99m: Remember that this guy is trapped but not organised. Background levels are higher because only 1-5% of the tracer is taken up by the thyroid gland. A common scenario to choose Tc over Iodine, is when they've had a recent thyroid blocker on board (iodinated contrast is the sneaky one).

Random Trivia on Breast Feeding (they love this shit):

- * *Tc-99m*: You can resume breast feeding in 12-24 hours
- * *I-123*: You can resume breast feeding in 2-3 days
- * *I-131*: You should not breast feed - pump and dump.

Iodine Uptake Test:

You give either 5 milli Ci of ¹³¹I or 10-20 milli Ci of ¹²³I. This is conventionally reported at 4-6 hours, and 24 hours. Normals are 6-18% (4-6 hours), and 10-30% at 24 hours. A correction for background is done on measurements prior to 24 hours (using the neck counts - thigh counts).

Factors affecting the test

- Renal Function (increases stable iodine pool, reduces numbers)
- Dietary Iodine - variable and controversial
- Medications - thyroid blockers, Nitrates, **IV Contrast**, **Amiodarone**

Increased Uptake	Decreased Uptake
Graves	Primary or secondary causes of Hypothyroidism
Early Hashimoto	Renal Failure
Rebound after Abrupt withdrawal of antithyroid medication	Medications (<i>thyroid blockers, Nitrates, IV Contrast, Amiodarone</i>)
Dietary Iodine Deficiency	Dietary Iodine Overload

Graves Disease: About 75% of the time, if you have hyperthyroidism the cause is going to be graves. Graves is an autoimmune disease where an antibody to the thyrotropin receptor stimulates the thyroid to produce hormone. TSH will be very low, where T3 and T4 will be high. The classic clinical scenario is a middle aged women with a protracted course, pre-tibial edema, and exophthalmos. Scintigraphy is going to give you a homogeneously increased gland, with uptakes increased at both 4 hours and 24 hours. Sometimes the 24 hour uptake is lower than the 4 hours (or even at a normal range) - this is from rapid thyroid hormone production.



Visualization of the pyramidal lobe: The pyramidal lobe is seen in about 10% of normal thyroids. In patient's with Graves disease it is seen as much as 45% of the time. Therefore, it's suggestive when you see it.

Multi-nodular Toxic Goiter (Plummer Disease): The classic scenario is an elderly women with weight loss, anxiety, insomnia, and tachycardia. The gland is typically heterogeneous, with uptake that is only moderately elevated. The nodules will be hot on the background of a cold gland.

Toxic Multi-nodular Goiter vs Non-Toxic Multi-nodular goiter: The toxic goiter will have hot nodules on a background of cold thyroid. The Non-toxic one will have warm/hot nodules on a normal background of the thyroid.

Graves	Toxic Multi-Nodular Goiter
Uptake High :70s	Uptake Medium High: 40s
Homogenous	Heterogeneous

Hashimotos - The **most common cause of goitrous hypothyroidism** (in the US). It is an autoimmune disease that causes hyper first then hypothyroidism second (as the gland bums out later). It's usually hypo - when it's seen. It has an **increased risk of primary thyroid lymphoma**. Step 1 trivia; associated with autoantibodies to thyroid peroxidase (TPO) and antithyroglobulin. The appearance of the hypothyroid gland is typically an inhomogeneous gland with focal cold areas. The hyperthyroid (acute) gland looks very much like Graves with diffusely increased tracer.

Subacute Thyroiditis: If you have a viral prodrome followed by hyperthyroidism, and then thyroid uptake scan shows a **DECREASED %RAIU you have de Quervains** (Granulomatous thyroiditis). During this acute phase, the disease can mimic Graves with a low TSH, high T3 and high T4. The difference is the uptake scan. After the gland bums out, it may stay hypothyroid or recover. If they ask you about this, it's most likely going to try and fool you into saying Graves based on the labs, but have a low % RAIU.

Hot Nodule vs Cold Nodule: Most thyroid nodules are actually cold, and therefore most are benign (colloid, cysts, etc..). In fact, cold nodules in a multi-nodular goiter are even less likely to be cancer compared to a single cold nodule. Having said that, cold nodules are much more likely to be cancer when compared to a functional (warm) nodule.

Discordant Nodule: This is a nodule that is HOT on Tc" but COLD on I¹²³. Because some cancers can maintain their ability to trap, but lose the ability to organify a hot nodule on Tc, it shouldn't be considered benign until you show that it's also hot on I¹²³.

Gamesmanship: Iodine vs Tc

A classic move is to show you a thyroid that will take up Tc, but NOT Iodine on 24 hour imaging. This can be from a couple of things: (1) congenital enzyme deficiency that inhibits organification, (2) a drug like propylthiouracil that blocks organification.

Now if they just show you an Iodine Thyroid with low uptake on 24 hours, this is de Quervains, or a burned out Hashimotos.

Radioiodine Therapy

I^{131} can be used to treat both malignant and non-malignant thyroid disease.

Cancer

Actual subtypes and pathology of thyroid cancer have been discussed at length in the endocrine chapter. However, just a few points that are relevant to discuss here. Papillary is the most common subtype (papillary is popular), and it does well with surgery + I^{131} . Medullary thyroid CA (the one the MENs get), does NOT drink the I^{131} and therefore doesn't respond well to radiotherapy. Prior treatment can also make you more resistant to treatment, and re-treatment dosing is typically 50% more than the original dose.

Things that make you treatment resistant:

- * Medullary Subtype CA (will not drink the tracer)
- * History or prior I^{131} ("easy gland has been killed off")
- * History of Methimazole treatment (even if years ago)

Medullary Subtype CA

Neuroendocrine in origin, so can occasionally (around 10%) have uptake on MIBG or Octreotide. They will be cold on thyroid scan and don't drink the treatment I^{131} .

Associated with MEN 2a and 2b

So, normally the patient gets diagnosed and then they go for surgery. After surgery they will come to nuclear medicine. You expect that they will have some residual thyroid (it's really hard to get it all out). Prior to actually treating them you will give them a tiny dose of I^{131} to see how much thyroid they have left. If the uptake is less than 5% this is ideal. Uptake more than 5% will result in a painful ablation (may need steroids on top of the NSAIDs) and may need to go back to the OR. Next, you will treat them. You want their TSH really ramped up. The higher the TSH the thirstier the cancer /residual thyroid tissue. An ideal TSH is like 50 (30 would be a minimum).

How do you get the TSH up?

- * There are two ways;
 - o (1) is to stop the thyroid hormone (post op they are obviously hypothyroid),
 - o (2) is to give recombinant TSH “Thyrogen.”

How do you decide on dosing?

- * Dosing is dependent on the stage of the disease; 100 for thyroid only, 150 for thyroid + nodes, 200 for distal. They are told about the precautions etc... Then you give them the dose. Before you let them go home, you test them to see if they need to go to the hospital.

So when do patients need to be admitted to the hospital?

- * NRC limit is 7mR/h measured at 1 meter from the patient’s chest (some agreement states use 5mR/h). The number to remember is **33 mCi of residual activity** (or 30mCi in some strict agreement states).

Possible Side Effects of Treatment:

- * Can cause **pulmonary' fibrosis if given to patient with lung mets**. This is really only the case of macro-nodular disease (as opposed to micronodular disease). That isn't necessarily a contraindication
- * Sjogrens have a greater risk of **salivary gland damage**
- * Salivary gland damage is dose related - so cancer treatment patients have a greater risk

What routes does the body use to eliminate I¹³¹?

- * Urine is the main way it is eliminated but sweat, tears, saliva, and breast milk are other routes.

If they don't need admitted to the hospital, what precautions should they take?

- * There is a whole bunch of crap they are asked to do. Drink lots of water (increase renal excretions). Suck on hard candy (keep radiotracer from jacking your salivary glands). Patients are encouraged to stay away from people (distance principal). Sleep alone for 3 days (no sex, no kissing). Good bathroom hygiene (flush twice, and sit down if you are a guy). Use disposable utensils and plates. Clothes and linens should be washed separately. Most of these things are done for 3 days.

Is it ok to breast feed? Is it ok to try and get pregnant?

- * No breast feeding. If you take I¹³¹ your breast feeding days are over (at least this time around).
- * No getting pregnant for at least 6-12 months after therapy

Other Trivia:

- * If you participated in the therapy, you need your thyroid checked 24 hours later.
- * If the patient got admitted to the hospital the RSO needs to inspect the room after discharge before the janitor can clean it or the next patient can move in.
- * Thyroglobulin is a lab test to monitor for recurrence. Anything over zero - after thyroidectomy, is technically abnormal, although the trend is more important (going up is bad)
- * **Severe uncontrolled thyrotoxicosis and pregnancy are absolute contraindications.**

*Gamesmanship - Iodine
Post Treatment*

If you see an Iodine Scan, and you see **uptake in the liver**, this is ALWAYS a post treatment scan.

Hyperthyroidism:

Classic Scenario - Patient is on dialysis and needs I^{131} Rx

Give I^{131} immediately following dialysis to maximize the time the I^{131} is on board.

Decrease dose as there is limited (essentially no) excretion until next dialysis.

Dialysate can go down sewer. Dialysis tubing needs to stay in storage.

I^{131} can also be used to treat hyperthyroidism. Dosing depends on the etiology; 15mCi for graves (more vascular), 30mCi for multi nodular (*harder to treat the capsule*). Again the TSH must be high for the therapy to be effective. By 3-4 months, there should be clinical evidence of resolution of signs and symptoms of hyperthyroidism, if I^{131} therapy was successful. As an aside, there is no such thing as an “emergent hyperthyroid treatment.” You can always use meds to cool it down. The standard medication is Methimazole. However, if there is an allergy to Methimazole, the patient is having WBC issues (side effect is neutropenia) or the patient is pregnant -use propylthiouracil (PTU). **PTU is recommended during pregnancy.**

What about Thyroid Eye Disease? It’s controversial, but **some people believe that thyroid eye disease will worsen after I^{131} treatment.** If you are prompted, I would just say have optho look at their eyes, bad outcome is likely severity related. You might not want to treat a bug eyed dude (depends on who you ask).

Wolff-Chaikoff Effect: Since we are talking about hyperthyroid treatment, there is no better time than to discuss the W.C. effect. Essentially, this is a reduction in thyroid hormone levels caused by ingestion of a large amount of iodine. The Wolff-Chaikoff effect lasts several days (around 10 days), after which it is followed by an "escape phenomenon." The W.C. effect can be used as a treatment principle against hyperthyroidism (especially thyroid storm) by infusion of a large amount of iodine to suppress the thyroid gland. The physiology of the W.C. effect also explains why hypothyroidism is sometimes produced in patients taking several iodine-containing drugs, including amiodarone.

Parathyroid Imaging

What Causes Hyperparathyroidism ?

- * **Most common cause is a hyperfunctional adenoma (85%).**
- * Second most common cause is multiple gland hyperplasia (12%).
- * Third most common cause is cancer (3%).

Nuclear medicine can offer two techniques to localize these lesions; dual phase, and dual tracer.

Dual Phase Technique

In dual phase technique, a single tracer (Tc^{99m}-Sestamibi) is administered, and both early (10 mins) and delayed (3 hours) imaging is performed. The idea is that sestamibi likes things with lots of **blood flow, and lots of mitochondria**. Parathyroid pathology tends to have both of these things, so the tracer will be more avid early, and stick around longer (after the tracer washes out of normal tissue). SPECT can give you more precise localization.

Testable Trivia: Sestamibi parathyroid imaging depends on mitochondrial density and blood flow”

False Positives:

Caused by things other than parathyroid pathology that like to drink sestamibi.

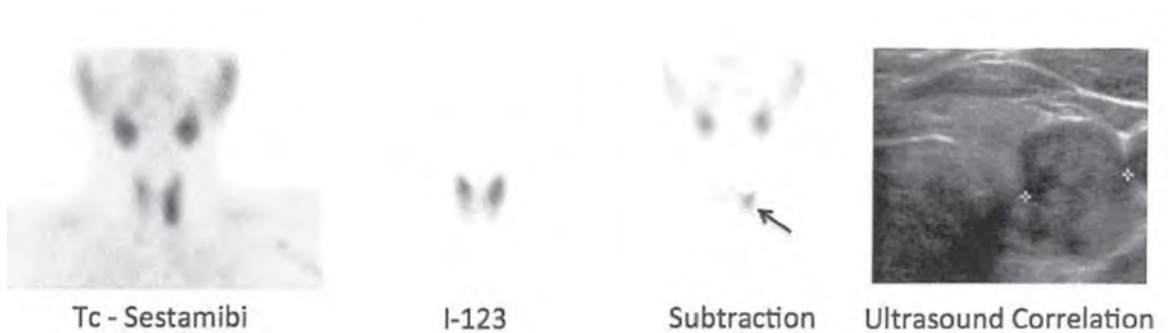
- * Thyroid Nodules
- * Head and Neck Cancers
- * Lymphadenopathy
- * Brown Fat

Dual Tracer Technique

In dual tracer technique two different agents are used and then subtraction is done. This first agent is chosen because it goes to both thyroid and parathyroid (options are either Tc⁹⁹-Sestamibi or 201- Thallium Chloride). The second agent is chosen because it only goes to the thyroid (options are either I-123 or Pertechnetate). When subtraction is done, anything left hot could be a parathyroid adenoma.

Problems:

- * Motion: subtraction imaging can't tolerate much motion
- * Stuff Messing with the Thyroid Tracers: recent iodinated contrast, etc...



Parathyroid Adenoma - Shown on Dual Tracer Method

Gamesmanship - MIBI - Lymph Nodes

On any study, parathyroid or a heart, if the tracer is MIBI than you should NOT see lymph nodes. If you see lymph nodes they are suspicious (maybe cancer). Next step would be ultrasound to further evaluate them.

Oh, and don't forget about focal breast uptake (also cancer), - Breast Specific Gamma Imaging (BSGI) uses MIBI for a reason.

CNS Imaging

The goal of brain imaging in nuclear medicine is to evaluate function (more than anatomy). Typically you are dealing with SPECT brain, FDG Brain, and Cisternograms / shunt studies.

SPECT

The idea behind brain SPECT is that you can look at brain blood flow, which should mimic metabolism. You have two main tracers **Tc HMPAO** (hexamethylpropyleneamine oxime) and **Tc ECD** (ethyl cysteinate dimer). HMPAO and ECD can be used in both dementia imaging and for seizure focus localization. The two tracers have similarities and differences, and the contrast between them lends well to multiple choice tests.

HMPAO	ECD
Neutral and Lipophilic	Neutral and Lipophilic
Accumulate in the cortex proportional to blood flow (Gray Matter > White Matter)	Accumulate in the cortex proportional to blood flow (Gray Matter > White Matter)
Washout is fast	Washout is slow (more rapid clearance from blood pool)
Uptake favors the frontal lobe, thalamus, and cerebellum	Uptake favors the parietal and occipital lobes * <i>Makes comparison between HMPAO and ECD difficult</i>

Key points:

- Both agents pass blood brain barrier and stick to gray matter proportional to CBF
- HMPAO washes out faster
- ECD washout is slower, has better background clearance, and does not demonstrate intracerebral redistribution.

Tc DTPA - This is another agent that can be used for flow.

Key Points:

- DTPA does NOT cross the blood brain barrier and therefore cannot be used for brain parenchymal imaging. *You can NOT do SPECT
- Has the advantage over HMPAO and ECD in that it can be repeated without delay

Dementia imaging patterns: *will be discussed in the FDG brain section below.*

Seizure Focus:

The goal of nuclear imaging regarding seizures is to attempt to localize a seizure focus (sometimes they do ok if they cut it out). The idea is that **a seizure focus will be hot** (hypermetabolic and hyperperfusion) **during the seizure “ictal.”** Then **cold between seizures “interictal.”** You need to inject tracer (HMPAO or ECD) within 30 seconds of the seizure to get a good study. PET can be used, but is less practical.

Thallium ²⁰¹

Thallium is produced in a cyclotron, decays via electron capture, and has a half life of around 3 days (73 hours). The major emissions are via the characteristic x-rays of its daughter products mercury 201 - at 69 keV and 81 keV. The tracer is normally given as a chloride and will therefore rapidly be removed from the blood

Thallium **behaves like potassium**, crossing the cell membrane by active transport (Na+/K pump). Tumors and inflammatory conditions will increase the uptake of this tracer. The higher the grade tumor, the more uptake you get. As Thallium requires active transport, it can be thought of as a viability marker - you need a living cell to transport it.

Normal Distribution: Thyroid, salivary glands, lungs, heart, skeletal muscle, liver, spleen, bowel, kidneys, and bladder. Any muscle twitching will turn hot.

If you are going to use it with Gallium, you must use the Thallium first as the Gallium will scatter all over the Thallium peaks.

High Yield Trivia / Uses:

- Toxoplasma Infection is Thallium Negative, Lymphoma is Thallium Positive
- Kaposi Sarcoma is Thallium Positive (Gallium Negative)
- Tumor is Thallium Positive, Necrosis is Thallium Negative

Tumor vs Necrosis:

The tracers used for SPECT tumor studies are different than those used for dementia or seizures. The tumor tracers are ^{201}Tl (more common) and $^{99\text{m}}\text{Tc}$ Sestamibi (less common). ^{201}Tl is a potassium analog, that enters the cell via the Na/K pump. Inflammatory conditions will increase the uptake of this tracer, but not as much as tumors. The higher the tumor grade, the more intense the uptake. Thallium can be thought of as a marker of viability, as it will localize in living tumor cells, and not necrosis. The control is the scalp (abnormalities will have greater uptake than the scalp). You can use Thallium in combination with perfusion tracers (HMPAO).

Tumor vs Necrosis	
Thallium Hot, HMPAO Cold	Tumor
Thallium Cold, HMPAO Cold	Necrosis

CNS Lymphoma vs Toxoplasmosis:

As discussed in the neuro chapter CNS lymphoma vs CNS Toxoplasmosis can be a diagnostic dilemma. **Thallium has a role in helping to distinguish the two (Toxo Cold, Lymphoma Hot).** Please refer to the neuro chapter for additional discussion.

Brain Death

You are looking for the presence (or absence) of intracerebral perfusion to confirm brain death. So that you don't keep Grandma around as a piece of broccoli, you need to have a tourniquet on the scalp - otherwise you might think scalp perfusion is brain perfusion and say she's still alive. **You have to identify tracer in the common carotid - otherwise the study must be repeated. In the setting of brain death, tracer should stop at the skull base. The hot nose sign,** is seen secondary to perfusion through the external carotid to the maxillary branches. As a point of trivia - the hot nose sign cannot be used to call brain death, it is a "Secondary Sign."



Brain Death - Hot Nose Sign

Stroke

There is no reason, ever, under any circumstances known to man, women, or beast to ever, ever use SPECT to diagnose stroke. Having said that, you can look at stroke with SPECT and will therefore likely be asked questions about it.

The big take home points are this:

- * *Acute Stroke is Cold*
- * *Sub Acute Stroke can be warm - from luxury perfusion (blood flow is more than dead cells need).*
- * *Chronic Stroke is Cold*

Ischemia (TIAs)

You can evaluate for cerebrovascular reserve by first giving acetazolamide (Diamox) - which is a vasodilator, followed by a perfusion tracer. Normally you should get a 3-4x increase in perfusion. However, **in areas which have already maxed out their auto regulatory vasodilation (those at risk for ischemia) you will see them as relatively hypointense.** These areas of worsening tracer uptake may benefit from some revascularization therapy.

FDG-PET

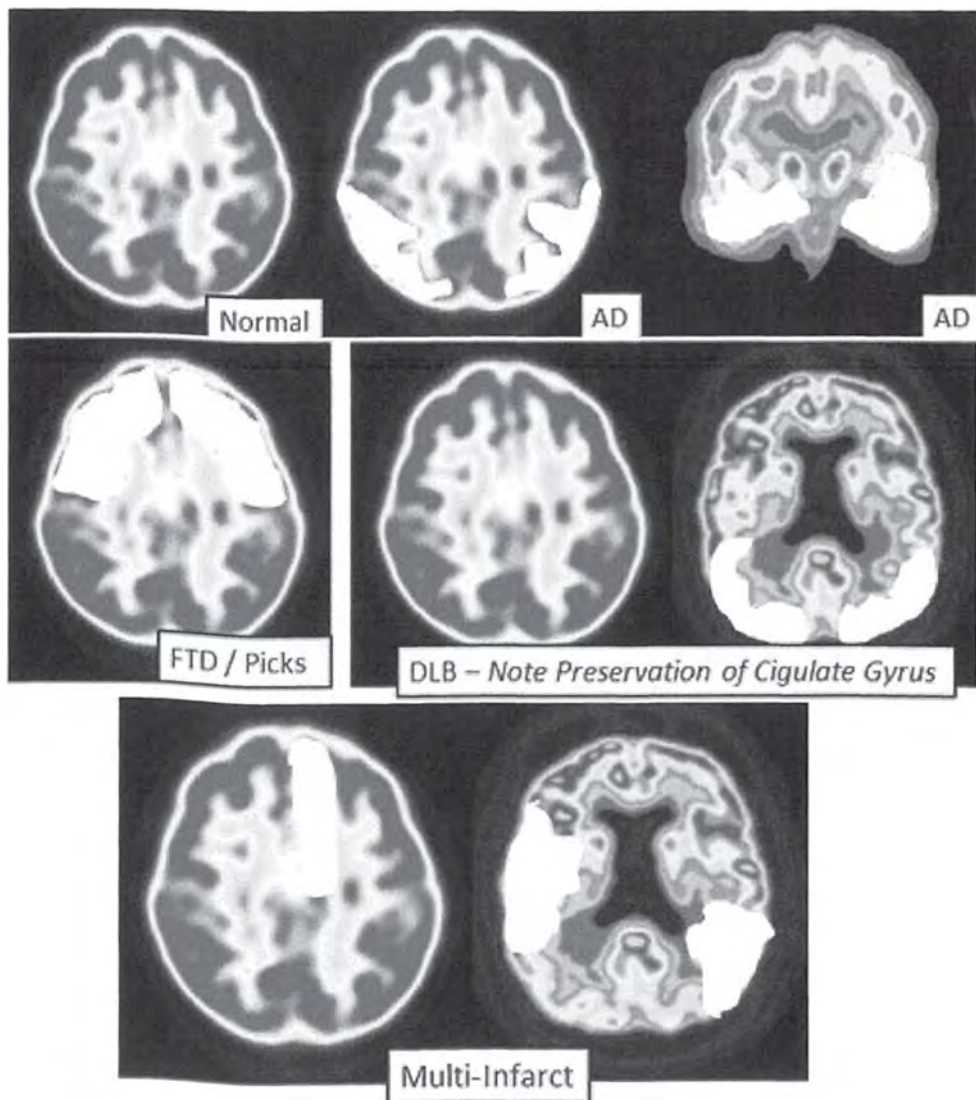
PET can assess perfusion ($^{15}\text{O-H}_2\text{O}$) but typically it uses ^{18}F FDG to assess metabolism (which is analogous to perfusion). Renal clearance of ^{18}F FDG is excellent, giving good target to background pictures. Resolution of PET is superior to SPECT.

It's important to remember that external factors can affect the results; bright lights stimulating the occipital lobes, high glucose (>200) causes more competition for the tracer and therefore less uptake, etc...

The most common indication for FDG Brain PET is dementia imaging. Because blood flow mimics metabolism HMPAO and ECD can also be used for dementia imaging and the patterns of pathology are the same.

Dementia is discussed in detail in the neuroradiology chapter. Please refer to the masterpiece that is the neuro chapter for additional details.

FDG PET - Brain		
Alzheimers	Low posterior temporoparietal cortical activity	Identical to Parkinson Dementia
Multi Infarct	Scattered areas of decreased activity	
Dementia with Lewy Bodies	Low in lateral occipital cortex	Preservation of the mid posterior cingulate gyrus (Cingulate Island Sign)
Picks / Frontotemporal	Low frontal lobe	
Huntingtons	Low activity in caudate nucleus and putamen	

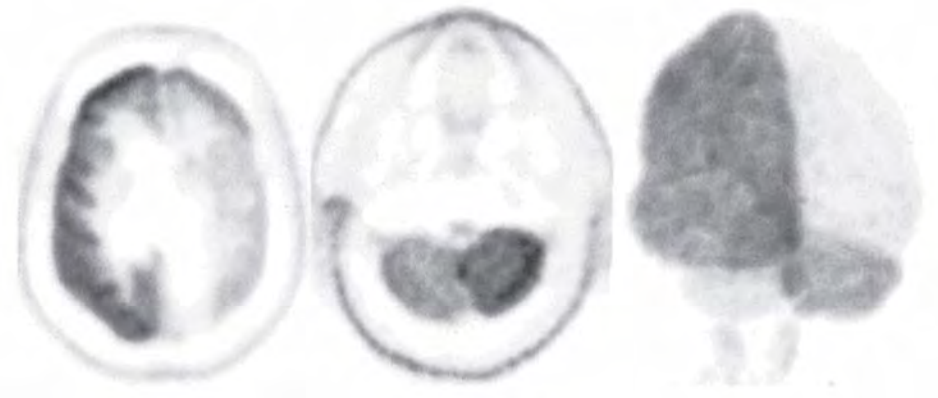


Miscellaneous Conditions For Which Pet Brain has Utility.

Crossed Cerebellar Diaschisis (CCD)

Depressed blood flow and metabolism affecting the cerebellar hemisphere after a contralateral supratentorial insult (infarct, tumor resection, radiation).

Creates an Aunt Minnie Appearance:



Crossed Cerebellar Diaschisis

CSF Imaging

The principle involved in imaging the CSF consists of intrathecal administration that will safely follow CSF and remains in the CSF compartment until it is absorbed through the conventional pathways. The **most common tracer used is ^{111}In - labeled DTPA**. So, you have to do an LP on the dude (it's intrathecal).

Normal Examination

- * Time Zero - You do the LP
- * 2-4 hours it ascends and reaches the basal cisterns
- * 4 hours - 24 hours it flows around the sylvian and interhemispheric cistern
- * At 24 hours it should clear from the basilar cisterns and be over the cerebral convexities

Abnormal Examination (general principles)

- * Tracer in the lateral ventricles
- * Failure to clear from the cisterns and localize over the convexities by 24 hours

Communicating Hydrocephalus: Normal pressure hydrocephalus is wet, wacky, and wobbly (incontinent, confused, and ataxic) clinically, and the “ventricular enlargement out of proportion to atrophy” on CT.

On scintigraphy you are looking for:

- * *Early entry (4-6 hours) of tracer into the lateral ventricles*
- * *Persistence of tracer in the lateral ventricle > 24 hours*
- * *Delay in Assent to the parasagittal region > 24 hours*



NPH - Persistent Tracer in the Ventricles > 24hours

Since radiotracer shouldn't normally enter the ventricles, a radionuclide cisternogram cannot be used to distinguish communicating from noncommunicating hydrocephalus. Historically (1930s) you could tell by injecting the material directly into the lateral ventricles.

This vs That: NPH vs Non obstructive (communicating) hydrocephalus. NPH will have a normal opening pressure on LP.

CSF Leak: You can use CSF tracers to localize a leak. The most common sites of CSF leak (fistulas) are between the cribriform plate and ethmoid sinuses, from the sella turcica into the sphenoid sinus and from the ridge of the sphenoid to the ear. The study is like a bleeding scan, in that the leak must be active during the test for you to pick it up.

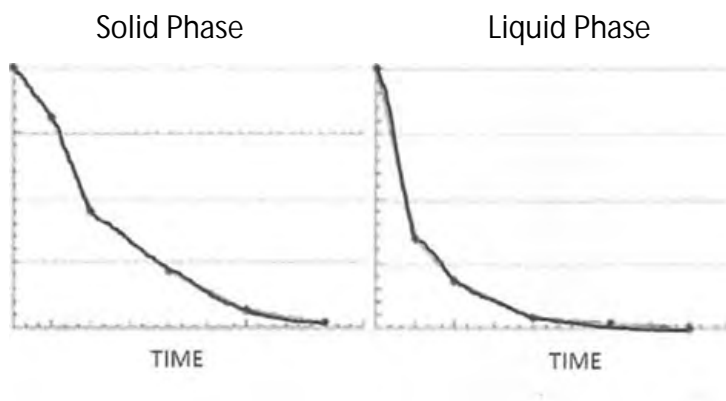
How is it done? You image around the time the CSF is at the basilar cisterns (1-3 hours) and also image pledgets (jammed up the nose prior to the exam). You compare tracer in serum to the pledgets (ratio greater than 1.5 is positive).

Shunt Patency: There are a bunch of ways to do this. Most commonly Tc labeled DTPA is used (^mIn - labeled DTPA could also be used). Usually, the tracer is injected straight into the tubing.

- * Normal Test will show tracer in the peritoneum - shows distal end is patent.
- * You can manually occlude the distal limb to force tracer into the ventricles - shows proximal end is patent.
- * If the tracer fails to reflux into the ventricles, or it does but then doesn't clear you can think proximal obstruction
- * If there is delayed tracer flow into the peritoneum (> 10 minutes = delayed), this can mean partial distal obstruction.

GI Imaging

Gastric Emptying : Believe it or not, this study is actually considered the “gold standard” to evaluate gastric motor function. The primary indication is typically gastroparesis (usually in a diabetic). The exam should be performed fasting (at least 4 hours). Some texts say that it should be done in the first 10 days of the menstrual cycle to prevent hormones from interfering (I’m sure this recommendation is evidence based). Most commonly Tc labeled sulfur colloid is used, on a standardized liquid meal, solid meal (egg whites), or both. **Solids are more sensitive**, but you can have emptying problems from liquids only and normal emptying from solids. The most likely test question is to understand the difference in curves between solids and liquids. The main point is that solids have a “**lag phase**” in which the stomach helps grind up the food into smaller parts (liquids don’t have this). Lag Time can be increased in diabetic patients.



Another possible question is that “**attenuation correction**” plays a role in calculation of emptying times, as movement from the back of the stomach to the front can increase counts due to attenuation.

Esophageal Transit: Used (rarely) in the evaluation of esophageal motility disorders. The supposed advantage is the ability to give quantitative information. The patient is made to fast overnight, then fed Tc-99 sulfur colloid. Dynamic imaging is performed and transit time and /or residual esophageal activity is measured.

GI Bleeding: The goal of a GI bleeding scan is to localize the bleed (not to say there is one). Bleeding scan is sensitive to GI bleed rates as low as at 0.1ml/min (Mesenteric angiography, requires 1-1.5 ml/min bleeding).

Key Point:

- * *GI bleed scan detection -0.1 ml/min*
- * *Angiogram detection = 1.0 ml/min*

First Some Technical Stuff (Very Boring and High Yield)

Before the Tc-99 can be tagged to a RBC (*beta chain of the hemoglobin*) it must first be reduced. This is accomplished with stannous ion (tin). This is referred to as “tinning.”

There are 3 methods:

RBC Tagging

In Vivo

1. Tin (stannous ion) is injected into the patient
2. Then Tc-99m pertechnetate is injected
3. Tin binds to the hemoglobin then reduces the Tc (which then binds)

Although the process is super simple, you only get about 60-80% of it bound. So you have a lot of free Tc and a dirty image (poor target to background). Sometimes it fails miserably (via drug interaction - **heparinized tubing, or recent IV contrast**). The images are too crappy for cardiac wall motion studies, but can work for GI bleeding.

In Vivo - In Vitro (Modified Method)

1. Tin (stannous ion) is injected into the patient
2. After 15-30 mins you pull 3-5 cc of blood out of an IV line into a syringe with both Tc-99m pertechnetate and an anticoagulant
3. It's then re-injected 10 mins later

This one does a little better, binding close to 85%. Drug interactions (like heparin) are the most common cause of failure.

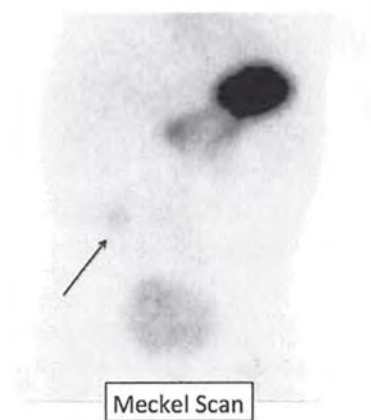
In Vitro

Blood is withdrawn and added to a kit with both Tin (stannous ion) and Tc. It's then re-injected. This method works the best (98% binding), but is the most expensive.

Reading the Study: You are looking for the appearance of tracer (outside the vascular distribution) that moves like bowel (can be antegrade or retrograde). You can get faked out by a lot of stuff; renal or bladder excretion (possibly with hydro), transplant kidney (classic trick - but again it won't move), varices or angiodysplasia (these shouldn't move), a penis with blood in it (this will look like a penis), hemangioma (this will be over the liver or spleen - and not move), and the last trick - Free Tc in the stomach. If you see gastric uptake* next look at the salivary glands and thyroid to confirm it's free Tc, and not an actual bleed.

Alternative (Stone Age) Way of Doing A Bleeding Scan; Back when dinosaurs roamed the earth, they used to do bleeding scans with Tc Sulfur Colloid. This had a variety of disadvantages including; fast clearance (had to do scan in 30mins), multiple blind spots (the stomach, splenic flexure, and hepatic flexures - as sulfur colloid goes to the liver and spleen normally). The only possible advantages are that it requires less prep and has good target to background.

Meckel Scan: The Meckel Diverticulum is a remnant of the omphalomesenteric duct located near the distal ileum. These things can have ectopic gastric mucosa and present with painless bleeding in the pediatric population. Pertechnetate is used because it is taken up by gastric mucosal cells. So you are looking for tracer uptake in the pelvis (usually RLQ) around the same time as the stomach.



Only about 10-30% of meckels diverticulum will have gastric mucosa (these are the ones more likely to bleed).

Here are the Tricks:

- * You need to do the study when the patient is NOT bleeding (if they are bleeding - then do a bleeding scan).
- * *Pre-Treatment:* You can use a bunch of different stuff to make the exam better:
 - o Pentagastrin - enhances uptake of pertechnetate by gastric mucosa (also stimulated GI activity)
 - o H2 Blockers (Cimetidine and Ranitidine) block secretion of the pertechnetate out of the gastric cells making it stick around longer,
 - o Glucagon - slows gastric motility.
- * *False Positive:* Can occur from bowel irritation (recent scope, laxative use)
- * *False Negative:* Recent In vivo labeling or RBCs , Recent Barium Study (attenuated)

HIDA Scan:

Function and integrity of the biliary system can be evaluated for by using Tc-99m labeled tracers that mimic bilirubin's uptake, transport, and excretion. All the tracers are basically analogs of this iminodiacetic acid stuff.

Trivia: You need higher doses of tracer if the patient has hyperbilirubinemia

Prep for the test is diet control. You need to have not eaten within four hours (so your gallbladder is ready to fill), and have eaten within 24 hours (so your gallbladder isn't so full, it can't let any tracers in). If you haven't eaten for over 24 hours, then CCK can be given.

Normally, the liver will have prompt tracer uptake (within 5 minutes), then you will have excretion into the ducts, then the bowel - pretty much the same time you see the gallbladder. If the gallbladder is sick (obstructed), it will still not have filled within 60 min. This is the basic idea.

Acute Cholecystitis: Almost always (95%) patients with acute cholecystitis have an obstructed cystic duct. If you can't get tracer in the gallbladder within 4 hours, this suggests obstruction.

Rim Sign - A curved area of increased activity along the gallbladder fossa (hot rim, or pericholecystic hepatic activity sign) suggests a more angry gallbladder - sometimes gangrenous.



Hot Rim Sign - Hyperemia in Fossa

Mechanism of the Rim Sign: The mechanism is the result of inflammation causing regional hepatic hyperemia, with more radiopharmaceutical being delivered to this area of hepatic parenchyma;

Cystic Duct Sign - This sign is seen with acute cholecystitis. The sign describes a nub of activity in the cystic duct, with the remaining duct obstructed.

Chronic Cholecystitis: This can be shown two ways; (1) delayed filling of the GB (not seen at 1 hour, but seen at 4 hours), or (2) with a low EF (< 30%) with CCK stimulation. A reduced EF can also be seen in acute acalculous cholecystitis

Testable Trivia

- The Dose of CCK: 0.02 microgram/kg over 60 mins
- The Dose of Morphine 0.02-0.04 mg/kg over 30-60 mins

Biliary Obstruction: A lack of visualization of the biliary tree “Liver Scan Sign” can be seen with acute obstruction of the CBD.

Things that can go wrong:

- * No Bowel Activity , Persistent Blood Pool = Hepatocyte Dysfunction (Hepatitis)
- * No Bowel Activity, Blood Pool Goes Away Normally = Common Duct Obstruction
- * No Gallbladder Activity x 4 hours (or 1 hour + morphine) = Acute Cholecystitis
- * Abnormal GB emptying (EF < 30%) = Chronic Cholecystitis

Prompt Uptake - With Delayed Excretion (Medication)

Classically Dilantin (Chlorpromazine) and birth control pills can cause prompt tracer uptake and delayed clearance. This can mimic biliary obstruction

Biliary Atresia vs Neonatal Hepatitis

If you see a hepatobiliary scan (HIDA) in a kid, for sure this is the indication. Apparently, these two things are hard to tell apart clinically. If you see tracer in the bowel it's hepatitis, but just remember that it might be slow so you need super delays (24 hours if necessary). If you don't see it in the bowel, you might still need to repeat the study if you didn't charge up those hepatocytes with some phenobarb (up regulates the cytochrome system). In other words, a lot of places pre-medicate with phenobarbital to increase the utility of the test. So, if you operate early (kasai procedure) they do a lot better, so it's important not to screw this up.

Bile Leak

You can use HIDA tracer after trauma or surgery to look for bile leak. The trick is that you need delayed images, and look in the right paracolic gutter / pelvis. You can get tracer in the gallbladder fossa, mimicking a gallbladder.

Reappearing Liver Sign - Labeled bile may track superiorly into the perihepatic space and coat the surface of the liver. This can give the appearance of paradoxically increasing activity in the liver after an initial decrease in activity from liver emptying into the bowel.

Sulfur Colloid Liver Scan

Not frequently done because of the modern invention of CT. Sulfur Colloid tagged with Tc is quickly eaten by the liver's reticuloendothelial system. It can be used to see "hot" and "cold" areas in the liver. Classically the multiple choice question is Focal Nodular Hyperplasia Hot on sulfur colloid (although in reality it's only hot 30-40% of the time).

Sulfur Colloid Liver Scan	
Hepatic Adenoma	COLD
FNH	40% HOT, 30% COLD, 30% Neutral
Cavernous Hemangioma	COLD (<i>RBC Scan HOT</i>)
HCC	COLD, (<i>Gallium HOT</i>)
Cholangiocarcinoma	COLD
Mets	COLD
Abscess	COLD (<i>Gallium HOT</i>)
Focal Fat	COLD (<i>Xe HOT</i>)

Particle size is worth discussing briefly. Particles for this scan need to be 0.1 - 1.0 micrometers. This is the right size for the liver to eat them. If they are too big the spleen will eat them, and if they are too small the bone marrow will eat them. Also, realize that if they were too big they would get stuck in the lungs like a VQ on the first pass through.

Colloid Shift- In a normal sulfur colloid scan, 85% of the colloid is taken up by the liver (10% spleen, 5% bone marrow). In the setting of diffuse hepatic dysfunction, portal hypertension, hypersplenism, or bone marrow activation you can see change in uptake - shift to the spleen and bone marrow. The most specific causes of colloid shift are cirrhosis, diffuse liver mets, diabetes, and blunt trauma to the spleen.

Diffuse Pulmonary Activity - This is not normal localization of sulfur colloid. This is non-specific and can be seen with a ton of things (most commonly diffuse liver disease), but the first thing you should think (on multiple choice) is excess aluminum in the colloid. It can also be seen in primary pulmonary issues (reflecting phagocytosis by pulmonary macrophages).

Renal Activity on Sulfur Colloid = The most common cause is CHF (*maybe due to decreased renal blood flow and filtration pressure*). Alternatively, in the setting of renal transplant - this can indicate rejection *{due to colloid entrapment within the fibrin thrombi of the microvasculature}*. Other more rare causes include coxsackie B viral infection, disseminated intravascular coagulopathy, and thrombotic thrombocytopenic purpura.

Hemangioma Scan

This can be done using Tc labeled RBCs. Delayed blood pool is typically done (30 mins - 3 hours). If it's small (< 2cm) you'll need SPECT to localize it. Otherwise planar imaging will show a hot focus. You want to see marked HOT on delays, with no real hot spot on immediate flow or immediate pool. Angiosarcoma could be HOT on delays but would also be hot on flow. A partially fibrosed hemangioma may be a false negative.

Spleen Scan

You can use heat damaged Tc labeled RBCs to localize to the spleen. A possible indication might be hunting ectopic spleen.

GU Imaging

Imaging of GU system in nuclear medicine can evaluate function (primary role), or it can evaluate structure.

Function (dynamic):

Normal kidney function is 80% secretion and 20% filtration. Tracer choice is based on which of these parameters you want to look at.

Tc-DTPA: Almost all filtration and therefore a great agent for determining GFR. A piece of trivia is that since a small (5%) portion of DTPA is protein bound (and not filtered) you are slightly underestimating GFR. Critical organ is the bladder.

Tc-MAG 3: This agent is almost exclusively secreted and therefore estimates effective renal plasma flow (ERPF). It is cleared by the proximal tubules. Critical organ is the bladder.

Tc GH (glucoheptonate): This agent can be used for structural imaging (discussed later in this section), or functional imaging as it is filtered. Critical organ is the bladder.

Tc DTPA	Tc MAG 3	Tc GH
Filtered (GFR)	Secreted (ERPF)	Filtered
Good For Native Kidneys with Normal Renal Function	Concentrated better by kidneys with poor renal function	Good for dynamic and cortical imaging.
Critical Organ Bladder	Critical Organ Bladder	Critical Organ Bladder

There are essentially 5 indications for dynamic (functional) scanning: (1) Differential Function (2) suspected obstruction, (3) suspected renal artery stenosis, (4) Suspected Complication from Renal Transplant, (5) Suspected Urine Leak.

Some basics:

Images are obtained posteriorly (anterior if patient has a transplant or horseshoe). Typically dynamic exams have 3 phases; blood flow phase, cortical phase, and clearance phase.

Differential Function

This is a basic exam with the standard flow, cortical, and clearance phases.

Flow: Begins within 20 seconds of injection. Flow will first be seen in the aorta. Then as it reaches the renal arteries, the kidneys should enhance symmetrically and about equal to the aorta (at that time).

Flow	
Decreased (symmetric)	Technical Error - poor bolus
Decreased (asymmetric)	Renal Artery Thrombosis. Renal Vein Thrombosis. Chronic High Grade Obstruction. Acute Rejection. Acute Pyelonephritis.
Increased (asymmetric)	Renal Artery Aneurysm

An important piece of trivia is that ATN, Interstitial Nephritis, and Cyclosporin toxicity will all have normal perfusion/flow.

Cortical (parenchymal): This is the most important portion of the exam (with regard to differential function). An area of interest is drawn around the kidneys and a background area of interest is also drawn (to correct for the background). This can be screwed up by drawing your background against the liver or spleen (which is not true background since they will take up some tracer). You want to measure this at a time when the kidney is really drinking that contrast, but not so late that it is putting it in the collection system. Most places use around 1 min. A steep slope is good.

Clearance (excretory): Radiotracer will begin to enter the renal pelvis, collecting system, and bladder. In a normal patient, you will be down to half peak counts at around 7-10 mins. If you wanted to quantify retention of tracer you could look at a 20/3 or 20/peak ratio.

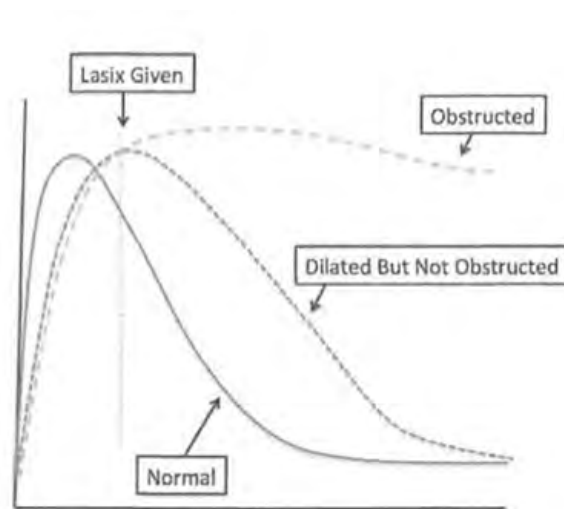
20/3 or 20/peak ratio: This is a method of quantifying retention of radiotracer by comparing the peak count at 20 minutes with the peak count at 3 mins (normal < 0.8) or the peak count (normal 0.3).

Suspected Obstruction (“The Lasix Renogram”)

The exam is performed the same as a standard dynamic exam (blood flow, cortical, and clearance), with a 30 minute wait after clearance. If there is still activity in the collecting system, a challenge is performed with Lasix. The idea is that a true obstruction will NOT respond to the Lasix, whereas a dilated system will empty when overloaded by Lasix. The study can be done with MAG-3 or DTPA. Mag-3 does better with patients with poor renal function, and thus is used more commonly.

The exam is interpreted as follows:

- * No obstruction = tracer clears from collecting system without need for Lasix
- * No obstruction = Washout of 50% of the tracer within 10 minutes of Lasix administration
- * Indeterminate = Washout of 50% of the tracer within 10-20 minutes of Lasix administration
 - o The most common cause for this indeterminate result is a very dilated pelvis and subsequent “reservoir effect.”
- * Obstructed= Washout taking longer than 20 mins after Lasix administration



Source of False Positive for Obstruction:

- * Poor response to Lasix - secondary to bad renal function, or dehydration at baseline
- * “Reservoir Effect” - very dilated renal pelvis, delaying transit time
- * Back Pressure Effects - Full or Neurogenic Bladder can generate back pressure and not let the kidneys empty (can be resolved with a foley catheter).

Suspected Renal Artery Stenosis (“Captopril Renogram”)

The study can be performed in one of two ways (both using MAG 3 as the typical tracer). The first is a standard dynamic study, followed by ACE inhibitor. The second as a baseline study with V_2 dose, followed by a full dose of ACE inhibitor. A “normal study” will occur if there is no difference between the baseline and the captopril studies.

The appearance of RAS will vary depending on the tracer given:

- * DTPA - Remember this is a GFR tracer. A sick kidney will have decreased uptake and flow, because of loss of perfusion pressure.
- * MAG 3 - Remember this is a Secreted tracer. A sick kidney will have marked tracer retention, with a curve similar to obstruction.

If it's bilateral up or bilateral down, it's not RAS. If the baseline study has asymmetrically poor function, that isn't positive for RAS, you need to see it worsen ($> 10\%$).

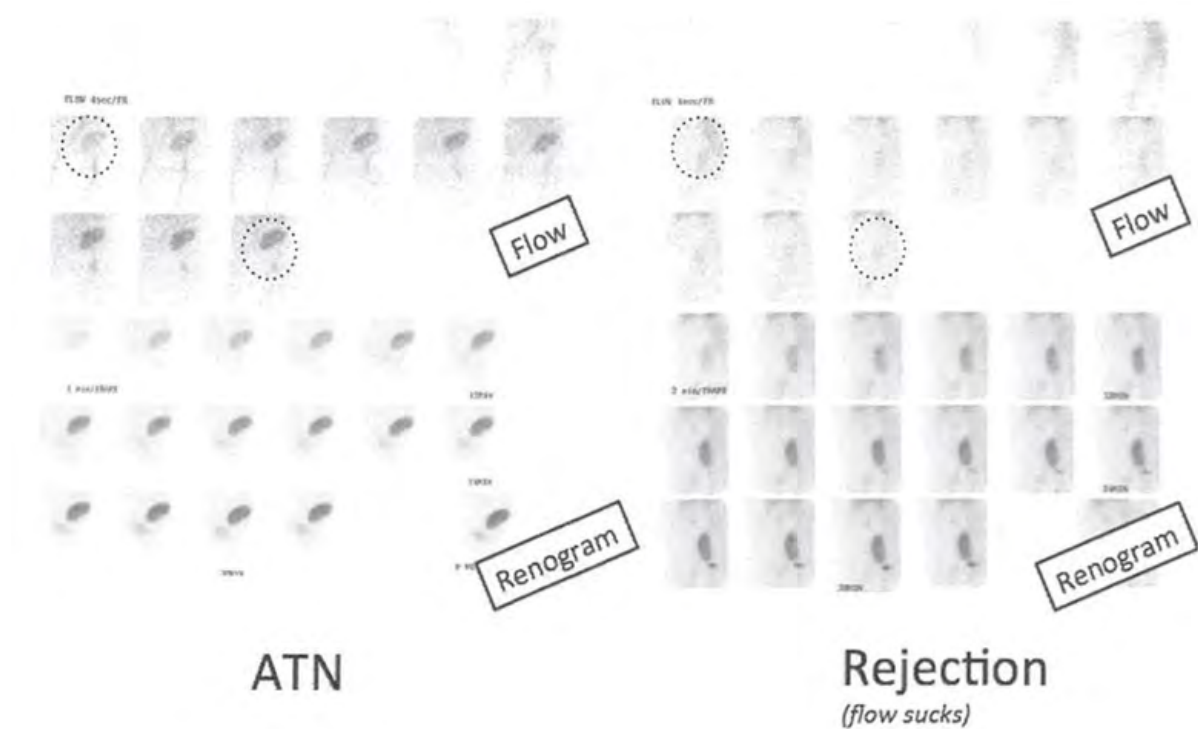
Trivia related to ACE inhibitor administration; they need to stop their ACE inhibitor prior to the renal study (3-5 days if captopril). They should be NPO for 6 hours prior to the test (for PO ACE inhibitors).

Suspected Complication from Renal Transplant

ATN vs Rejection: The most common indication for nuclear medicine in the setting of renal transplant is to differentiate rejection from ATN. ATN is usually in the first week after transplant, and is more common in cadaveric donors. There will be preserved renal perfusion with delayed excretion in the renal parenchyma (elevated 20/3 ratio, delayed time to peak). ATN usually gets better. There is an exception to this rule, but it will confuse the issue with respect to multiple choice, so I'm not going to mention it. Cyclosporin toxicity can also look like ATN (normal perfusion, with retained tracer) but will NOT be seen in the immediate post op period. Rejection will have poor perfusion, and delayed excretion. A chronically rejected kidney won't really take up the tracer.

In a Normal DMSA or ATN (with mag3 tracer), the nephrographic appearance is the same. Tracer in the cortex, and that's it.

ATN	Immediate Post OP (3-4 days post op)	Perfusion Normal	Excretion Delayed
Cyclosporin Toxicity	Long Standing	Perfusion Normal	Excretion Delayed
Acute Rejection	Immediate Post OP	Poor Perfusion	Excretion Delayed



Fluid Collections: Fluid collections seen after a transplant include urinomas, hematomas, and lymphoceles. All 3 can cause photopenic areas on blood pool imaging.

- * Urinoma: Usually found in the first 2 weeks post op. Delayed imaging will show tracer between the bladder and transplanted kidney. A hematoma is not going to have tracer in it.
- Lymphocele: Usually found 4-8 weeks after surgery. The cause is a disruption of normal lymphatic channels during perivascular dissection. Most are incidental and don't need intervention. If they get huge, they can cause mass effect. This will look like a photopenic area on the scan.

Vascular Complications: Both arterial and venous thrombosis will result in no flow or function. If you suspect renal artery stenosis (most common at the anastomosis), you can do a captopril study, with results similar to RAS if there is a stricture.

Structure

If you want to look at the renal cortex, you will want to use an agent that binds to the renal cortex (via a sulfhydryl group). You have two main options with regard to tracer.

- **Tc-DMSA:** This is the more commonly used tracer. It binds to the renal cortex and is cleared very slowly. **Critical organ is the kidney** (*notice other renal tracers have the bladder as their critical organ*).

- **Tc GH (glucoheptonate):** This is less commonly used and although it binds to the cortex, it is also filtered and therefore can be used to assess renal flow, the collecting system, and the bladder. Critical organ is the bladder.

DMSA is the preferred cortical imaging agent in pediatrics, because it has a lower dose to the gonads (even though its renal dose is higher than TcGH).

Indications for the exam:

- *Acute Pyelonephritis: Can appear as (a) focal ill defined area of decreased uptake, (b) multifocal areas of decreased uptake, (c) diffuse decreased uptake - in an other-wise normal kidney.*
 - o Scarring and Masses can also appear as focal areas of decreased uptake - although scarring usually has volume loss.*
- * *Column of Bertin vs Mass: Simply put, the mass will be cold. The Column of Bertin (normal tissue) will be take up tracer.*

*The trick on DMSA is just like reading CXR or Chest CT-you need to know if it is acute or chronic. The clinical history changes your DDx. **

- *Defects on DMSA with acute renal problems = pyelo.*
- *Defects on DMSA with chronic renal problems = scar (or mass).*

Testicular

This hasn't been done in the United States since 1968, therefore it is very likely to be on the test. The study is basically a blood flow study. The primary clinical question is testicular torsion vs other causes of pain (epididymitis). The tracer used is sodium pertechnetate ($\text{Na}^{99\text{m}}\text{TcO}_4$). Oh, don't forget to tape the penis out of the way - tape it up, not down like is required for the male residents on mammography rotations.

- * Normal - Symmetric low level flow to the testicles.
- * Acute (early) Torsion - Focal absence of flow to the affected side ("nubbin sign").
- * Delayed (late) Torsion - Sometimes called a missed torsion. The appearance is a halo of increased activity, with central photopenia.
- * Testicular Abscess - Identical to delayed torsion - halo of increased activity, with central photopenia.
- * Acute Epididymitis- Increased flow and blood pool to the affected side.

FDG PET (for cancer)

As mentioned before ^{18}F -FDG is cyclotron produced, and decays via positive beta emission to ^{18}O . The positron gets emitted, travels a short distance, then collides with an electron producing two 511 keV photons which go off in opposite directions. The scanner is a ring and when the two photons land 180 degrees apart at the same time the computer does math (which computers are good at) to localize the origin.

A CT component is fused over the PET portion. This is done for two reasons:

- (1) Anatomy - so you can see what the hell you are looking at.
- (2) Attenuation Correction - Dense stuff will slow down the photons, and the CT allows for correction of that. It also leads to errors, the classic one being a metallic pacemaker look bright hot on the corrected image (the computer overcorrected). This is a classic question. The answer is look at the source images (uncorrected).

Some other technical trivia is that FDG enters the cell via a GLUT 1 transporter and is then phosphorylated by hexokinase to FDG-6-Phosphate. This locks it in the cell. Normal bio distribution is brain, heart, liver, spleen, GI, blood pool, salivary glands, and testes. The collecting system and bladder (target organ) will also be full of it, because that's where it's getting excreted.

Variable areas of uptake: Muscles (classic forearm muscle uptake in the nervous chair squeezing patient). Breast and ovaries in females at certain stages of the menstrual cycle. Thymus in younger patients. Lastly, brown fat around the neck, thorax, and adrenals (especially in a cold room).

Ways to minimize brown fat uptake: Keep the room warm. Medications like benzodiazepines or beta blockers.

Tumors that are PET COLD	Not Cancer but PET HOT
BAC (Adeno In Situ) - Lung Cancer	Infection
Carcinoid	Inflammation
RCC	Ovaries in Follicular Phase
Peritoneal Bowel/Liver Implants	Muscles
Anything Mucinous	Brown Fat
Prostate	Thymus

Effects of Insulin and Blood Glucose

- * High Blood Glucose (> 150-200): The more glucose the patient has, the more competition is created for the FDG and you will have artificially low SUVs.
- * Insulin: So why not just give the patient some insulin??? It will drive it all into the muscles. This is a classic trick. PET with diffuse muscle uptake = Insulin Administration

Effects of being fat

- * Fat people will have HIGHER SUV values, because the fat takes up less glucose

When do you image? Following therapy an interval of 2-3 weeks for chemotherapy, and 8-12 weeks for radiation is the way to go. This avoids “stunning” - false negatives, and inflammatory induced false positive.

Who do you image? The main utility is extent of disease, and distal metastatic spread. Local invasion is tricky with a lot of things. Usually straight up CT or MR1 is better for local invasion and characterization.

What if you see the right ventricle ? The RV is not typically seen on PET unless it's enlarged. If you see the RV think about RVH.

Special Situations - Trivia

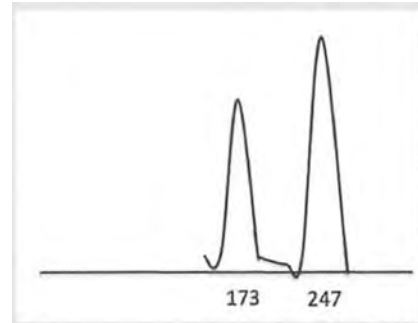
- Focal Thyroid Uptake - Requires Further Workup - might be cancer, might be nothing
- Diffuse Thyroid Uptake - Most often Autoimmune (Hashimoto) Thyroiditis
- RCC are COLD, Oncocytomas are HOT
- COLD Ground Glass Nodule = Cancer, HOT Glass Nodule = Infection
- The Reason HCC is often cold (60%) is that it has variable glucose-6-phosphatase and can't trap the FDG

Non - PET Cancer Imaging

In¹¹¹- Octreoscan

Indium¹¹¹

Indium is produced in a cyclotron and decays with a 67 hour half life via electron capture. It produces two photopeaks at 173 keV and 247 keV. Just like Gallium, In¹¹¹ in a liquid will carry a +3 valence and behaves like Fe⁺³, with the capability of forming strong bonds with transferrin.



The most common application is to bind it to WBC, although it can also be hooked to octreotide, or DTPA for CNS imaging (cisternography). Basically, you can hook indium to almost anything if you hook it first to a strong chelator like DTPA. As a point of trivia, you need to isolate the WBCs prior to labeling because the transferrin in the blood binds with greater affinity and will out-compete them.

¹¹¹In Pentetreotide is the most commonly used agent for somatostatin receptor imaging. The classic use is for carcinoid tumors, gastrinomas, paragangliomas, merkel cell tumors, lymphoma, small cell lung cancer, medullary thyroid cancer, and meningiomas.

I want to stress the random fact that Meningiomas take up Octreotide (and Tc MDP).

As a point of trivia, there are 5 octreotide receptors of which ¹¹¹In Pentetreotide can bind to two of them. The scan works because 80% of neuroendocrine tumors express these two receptors.



Meningioma – Hot on Octreotide and Tc MDP

Normal uptake is in the thyroid, liver, gallbladder, spleen, kidneys, bladder, and GI tract. Imaging is done in early and delayed phase. The advantage of the early phase (4 hours) is that the bowel activity is absent. The delayed is done to clarify that the abdominal tracer is of GI origin.

MIBG

MIBG is an analog of noradrenalin and is therefore taken up by adrenergic tissue.

MIBG is first line for tumors like pheochromocytoma, paraganglioma, and neuroblastoma. You can have MIBG with either I-123 or I-131. I-123 is better because it has better imaging quality. I-131 is cheaper, and the long half life allows for delayed imaging.

Tumors Showing I-131 MIBG Uptake

Paraganglioma (75%)
Pheochromocytoma (75-100%)
Carcinoid (70%)
Medullary Thyroid CA (10%)

Blocking the Thyroid Gland: The thyroid gland should be blocked, to prevent unintended radiation to the gland from unbound I-123 or I-131. This is accomplished with Lugol's Iodine or Perchlorate.

Medication Interaction with MIBG - High Yield Trivia

Certain medications interfere with the workings of MIBG and must be held. Medications include calcium-channel blockers, labetalol (other beta-blockers have no effect), reserpine, tricyclic antidepressants and sympathomimetics.

Biodistribution - Normal in liver, spleen, colon, salivary glands. The adrenals may be faintly visible. Note the kidneys are NOT seen.

Trivia: MIBG is superior to MDP bone scan for neuroblastoma bone mets. ****If you see a skeleton on MIBG the answer is diffuse bone mets.***

Gallium

Gallium can be used for tumors. Remember, it's very nonspecific with regard to infection, inflammation, or tumor. Classically, Gallium was used for lymphoma imaging.

Prostascint

¹¹¹In can be labeled to the antibody capromab Pendetide (Prostascint). Capromab is a monoclonal antibody which recognizes PSA antigen. Pendetide is the chelating agent. In my opinion, the exam is shit and doesn't work well.

When do you do the test? If you have a rising PSA and negative bone scan. The purpose of the study is to look for mets outside the prostate bed (soft tissue mets). If they do not have distal mets, they can be offered salvage therapy (radiation to the surgical bed). It's important to not obsess over the surgical bed, the real question is distal mets. Having said that the prostate bed is best seen on the lateral between the bladder and penis.

Testable Trivia: Prostascint will localize to soft tissue mets, NOT bone mets.

Sentinel Node Detection

A sentinel node is the node which receives afferent drainage directly from a primary lymph node. Surgeons want to know where these are at; especially with melanoma and breast cancer. The agent used for lymphoscintigraphy is 10-50nm Tc99m sulfur colloid.

Melanoma: Sentinel node mapping is done when you have a lesion between 1mm-4mm deep. Less than 1mm you are typically safe. More than 4 mm you are totally screwed and it makes no difference. Intradermal injection in 4 spots around the lesion / excision scar and imaging is done.

Breast Cancer: Cancer drains to the internal mammary chain nodes about 3% of the time. Knowing this, and which axillary node to go for first can help avoid aggressive lymph node dissection. Injections can be done superficial or deep (into the pectoral muscle).

Size Matters

Does particle size matter for sentinel node detection? Yes and No. Albumin is typically filtered, to a size of around 0.2 microns. The size is actually not essential for the test to be diagnostic (you could use the un-filtered liver spleen scan sulfur colloid in a pinch). The primary reason for this size is speed of exam. If you used larger particles you would be waiting all day for them to get to the lymph nodes.

Test	Particle Size
Lymphoscintigraphy	< 0.2 microns (< 200 nm)
VQ	10-100 microns
Liver Spleen	"Unfiltered" - so all sizes big and small

Breast Specific Gamma Imaging

Tc99 Sestamibi will concentrate in a breast cancer 6 times more than normal background breast tissue. It does pretty good, with the sensitivity supposedly near 90%. The technique is to give 20-30 mCi of Tc99 Sestamibi in the contralateral arm then image 20 mins later. A foot injection is often done if you are going to image both breasts. A dedicated gamma camera that can mimic a mammogram and provide compression.

Does Breast Density Affect Uptake / Distribution ?

Nope. The distribution is homogeneous regardless of density. Having said that, hormonal fluctuation can increase the background uptake.

When will background activity be lowest?

Around mid-cycle in premenopausal women.

What are some causes of false positive studies ?

Fibroadenoma, fibrocystic change, or inflammation can give a false positive

What are some causes of false negative studies?

Lesions that are small (< 1cm), or deep. Lesions located in the medial breast, and/or those overlapping with heart activity.

What about lymph nodes?

If you see lymph nodes on a “mibi” scan this is NOT normal, and they are concerning for mets.

Cardiac Imaging

Myocardial Perfusion / SPECT:

Tc Sestamibi and Tc Tetrofosmin are the most common tracers. They work by crossing the cell membrane and localizing in mitochondria (passive diffusion). They don't redistribute (like Thallium), giving better flexibility.

Sestamibi vs Tetrofosmin - Tetrofosmin is cleared from the liver more rapidly and decreases the chance of a hepatic uptake artifact.

Thallium - This is historical, with regard to cardiac imaging. It mimics potassium and crosses the cell membrane first by distribution related to blood flow - second by delayed redistribution (washout). Washout is delayed in areas with poor perfusion

Imaging Timing:

Tc studies (sestamibi and tetrofosmin) are done 30-90 mins after injection allowing for clearance from background

Thallium	Sestamibi and Tetrofosmin
Old	Newer
Crosses cell via Na/K pump	Crosses cell via passive diffusion (localizes in mitochondria)
Redistributes	Does NOT redistribute
Imaging must be done immediately after injection	Imaging typically done 30-90 mins after injection to allow for background to clear

Lung/ Heart Ratio: Only done with Thallium. If there is more uptake in the lungs, this correlates with multi-vessel disease or high grade LAD or LCX lesions.

General Principal: You will see less perfusion distal to an area of vascular obstruction (compared to normal myocardium). To improve sensitivity the heart is stressed. Under stress you need about 50% stenosis to see a defect (it needs to be like 90% without stress).

Preparation: Patient shouldn't eat for 4 hours prior to imaging (decreases GI blood flow). Patients should (ideally) stop beta-blockers, calcium channel blockers, and long acting nitrates for 24 hours prior to the exam - as these meds mess with the sensitivity of the stress portion. There are reasons to keep people on these meds (they might be getting risk stratification on medical therapy) - but I'd say for the purpose of multiple choice just know that those medication classes mess with stress imaging sensitivity.

Protocols: There are multiple ways to skin this particular cat. People will do two day exams; rest then stress. People will do one day exams stress then rest. The advantage to doing stress first is that you can stop if it's normal. Typically the dosing is low for the rest and high for the stress.

Chemical Stress: If you can't exercise, the modern trend is to give you Regadenoson - which is a specific adenosine receptor agonist. It's specific to a certain receptor having less bronchospasm than conventional adenosine or dipyridamole. If they get bronchospasm anyway you need to give them albuterol.

Findings:

Fixed Defect (seen on stress and rest)	Scar (prior infarct)
Reversible Defect (seen on stress, better on rest)	Ischemia
Fixed Defect with Reversible Defect around it	Infarct with peri-infarct ischemia
Transient Ischemic Dilation (LV cavity is larger on stress)	From diffuse subendocardial hypoperfusion producing an apparent cavity dilation. Correlated with high risk disease (left main or 3 vessel).
Fixed Cavity Dilation	Dilated cardiomyopathy -
Right Ventricular Activity on Rest	If has intensity similar to LV then think right ventricular hypertrophy
Lots of splanchnic (liver and bowel) activity	Means you aren't exercising hard enough - not shifting enough blood out of the gut.

Other Trivia:

Stunned v.v *Hibernating Myocardium:*

Stunned: This is the result of ischemia and reperfusion injury. It is an acute situation. The **perfusion will be normal**, but contractility will be crap. It will get better after a few weeks.

Hibernating: This is a more chronic process, and the result of severe CAD causing chronic hypoperfusion. You will have areas of **decreased perfusion and decreased contractility** even when resting (just like scar). Don't get it twisted, **this is not an infarct. This tissue will take up FDG more intensely than normal myocardium, and will also demonstrate redistribution of thallium.**

Rapid Review

Ischemia = Will take up less tracer (relative to other areas) on stress, and the same amount of tracer (relative to other areas) on rest. It's not normal heart so it won't contract well.

Scar = Won't take up tracer on rest or stress (it's dead). It's scar not muscle, so it won't contract normally either.

Stunned = The perfusion will be normal on both stress and rest, but the contractility is not normal.

Hibernating = Won't take up tracer on rest or stress (it's not dead, just asleep - like a bad soap opera plot). The difference between hibernating muscle and scar is that the hibernating muscle will take up FDG and redistribute thallium.

MUGA: This is an equilibrium radionuclide angiogram with cardiac pool images taken after the tracer has equilibrated to the intravascular space. This studies requires gating (the "G" in MUGA). The study is done using Tc 99 labeled RBCs, and the objective is to calculate an EF. Photopenic halo around the cardiac blood pool is a classic look for pericardial effusion. Regional wall motion abnormality on a resting MUGA is usually infarct (could be stunned or hibernating as well).

Most likely questions regarding MUGA:

- * **False Low EF:** Screwed up LAO view can cause overlap of LV with LA or RV or even great vessels - causing a false low EF. *
- * **False High EF:** Wrong background ROI (over the spleen), will cause over subtraction of background and elevate the EF.

Misc Trivia:

Rubidium 82: This is a potassium analog (mechanism is Na/K pump). This is similar to TI-201, and can be used as a similar agent. You can use it for PET myocardial perfusion, although it's not used in most places because of cost limitations. Also, because of the very short half life (75 seconds) it tends to give a dirtier image compared to PET of NH_3 .

I say made with a generator, you say Tc99 and Rubidium. * Rubidium is the only PET agent made like this, so that instantly makes it a testable fact.

Balanced Ischemia

Because rest and stress scintigraphy is essentially comparing one part of the heart to another it is susceptible to missing severe 3 vessel disease. In other words, what if the whole heart is abnormal? You wouldn't see any areas that are cold relative to the rest of the heart because the entire heart is cold.

So WTF do you do then? The answer is to look to a quantitative answer using PET perfusion agents (Rb^{82} and N^{13} Ammonia). This will tell you the coronary flow reserve (CFR).

Artifacts		
Breast Tissue “Soft Tissue Attenuation”	Decreased activity in anterior wall (may also affect septal and lateral - depending on body habitus)	Check for ECG changes and wall motion. If normal, then call it artifact. If not sure can repeat in prone position.
Left Hemidiaphragm “Soft Tissue Attenuation”	Decreased activity in inferior wall	Check for ECG changes and wall motion. If normal, then call it artifact.
Subdiaphragmatic Radiotracer Activity	Increased activity in inferior wall, can mask true defect. Can also mess with “normalization” of the ventricle and make the rest of the LV look low.	Liver Excretes Tc, so you see it in the liver and bowel. Little bit of exercise can be used to reduce GI blood flow.
Patient Motion (usually respiration)	Causes all kinds of problems	You can repeat because tracer is fixed for around 2 hours
Misregistration	Causes all kinds of problems	
Left Bundle Branch Block	Reversible or Fixed Septal Defects , sparing the apex	Seen more in exercise or dobutamine stress compared to vasodilators
Normal Apical Thinning	Normal variant	Look for matching stress and rest perfusion patterns with preserved wall function to show you this is normal (and not infarct)

Medication Trivia:

	Mechanism	Trivia
Dipyridamole	Inhibits the breakdown of adenosine - which builds up. Adenosine is a potent vasodilator.	No caffeine
Adenosine	Vasodilator	No caffeine; Side effects are worse than with dipyridamole. Rare side effect is AV block - which will get better when adenosine short half life runs out.
Regadenoson	Selective A ₂ A - causes less side effects	No caffeine
Dobutamine	Beta 1 agonist - acts like exercise by increasing heart rate and myocardial contraction.	Patient Can NOT be on a beta blocker. Best used in patient's who cannot have Adenosine or Dipyridamole. Better in patients with COPD or Asthma, or who have taken caffeine in the last 12 hours.
Aminophylline	Antidote for Adenosine	Half life is shorter than Dipyridamole - so must continue to monitor.

Nuclear Therapy

Treatment for Bone Pain:

There are currently three approved agents for bone pain associated with metastatic disease from breast and prostate cancer: (1) Sr^{89} -Chloride, (2) Sm^{153} EDTMP, and (3) Ra^{223} -dichloride.

Why does cancer cause bone pain / bone problems?

Metastatic disease leads to a tumor derived factor that increases osteolytic activity. You end up with increased fracture risk, osteopenia, and hypercalcemia of malignancy.

Can the patient get external radiation treatment with the therapy?

Yes, External radiation is not a contraindication and can be used with the therapy.

*Absolute contraindications (for Sr and Sm) include: Pregnancy, Breastfeeding, and renal Failure (<30GFR). Patient's with extensive bony mets (superscan) maybe shouldn't be treated either * controversial - arid therefore not likely tested.*

Sr^{89} (Metastron): It works by complexing with the hydroxyapatite in areas where bone turnover is the highest. It's the oldest, and worst of the three agents. It is a pure beta emitter. It has a high myelotoxicity, relative to newer agents and therefore isn't really used.

Sm^{153} (Quadramet): This is probably the second best of the three agents "*Samarium is a good Samaritan.*" It works by complexing with the hydroxyapatite in areas where bone turnover is the highest. It is a beta decayer. The primary method of excretion is renal. Unlike Sr^{89} about 28% of the decay is via gamma rays (103 kev) which can be used for imaging. Does have some transient bone marrow suppression (mainly thrombocytopenia and leukopenia), but recovers faster than Sr.

Sr^{89} (Metastron):	Sm^{153} (Quadramet)
15-30% drops in platelet and WBC from pre-injection	40-50% drops in platelet and WBC from pre-injection
8-12 weeks needed for full recovery	6-8 weeks needed for full recovery

Ra²²³ (Xofigo): This is the most recent of the three agents, and probably the best. The idea is that Ra²²³ behaves in a similar way to calcium. It is absorbed into the bone matrix at the sites of active bone mineralization. Its primary mechanism is the emission of 4 alpha particles, causing some serious double stranded DNA breaks.

Why is it the best?

- (1) It's an alpha emitter with a range shorter than Sr and Sm. This means less hematologic toxicity.
- (2) At least one trial actually showed a survival benefit in prostate CA.
- (3) It has a long half life (11.4 days) allowing for easy shipping.

What are the side effects?

Non-hematologic toxicities are generally more common than hematologic ones; diarrhea, fatigue, nausea, vomiting, and bone pain make the list.

Trivia: The general population is safe, as the gamma effects are low. Soiled clothing and bodily fluids should be handled with gloves, and clothes should be laundered separately. A 6 month period of contraception is recommended although none of this is evidence based (as per usual).

Sr ⁸⁹	Sm ¹⁵³	Ra ²²³
Pure Beta Emitter	Beta Emitter, with some imaggable gamma rays	Alpha Emitter
Most Bone Marrow Toxicity (longest recovery).	Less Bone Marrow Toxicity	Least Bone Marrow Toxicity
Renal excretion	Renal excretion	GI excretion
		Improves Survival (prostate mets)

Yttrium-90

This can be used as a radioembolization method, for unresectable liver tumors. This is a pure Beta emitter that spares most of the adjacent normal liver parenchyma as the maximum tissue penetration is about 10mm.

Prior to treatment with Y-90 the standard is to do a 99mTc MAA hepatic arterial injection. The primary purpose of this injection is to look for a lung shunt fraction. This fraction needs to be < 10% under ideal circumstances. You can still use Y-90 for 10-20% shunts but you need to decrease the dose. Above 20% the risk of radiation pneumonitis is too large.

Particle Size: The optimal particle size is between 20-40 um, as this allows particles to trap in the tumor nodules, but large enough to get stuck without totally obstructing. If you create a true embolization the process actually doesn't work as well because you need blood flow as a free radical generation source.

Radiation Dose: The dose is typically 100-1000 Gy delivered. The current thinking is that lesions require at least 70 Gy for monotherapy success.

Imaging: There are 175 Kev and 185 keV emissions you can use to image.

Trivia: The average half life is 2.67 days.

Radioimmune Therapy (RIT):

Monoclonal antibodies can be used with Indium¹¹¹ ibritumomab tiuxetan (Zevalin) for refractory non-Hodgkin lymphoma treatment, or as a first line treatment.

The idea is that you can give the antibody labeled with Indium¹¹¹ for diagnostic evaluation of the tumor burden and then if the bio distribution is ok you can give the antibody labeled with Y-90 for treatment.

Trivia: The antibody binds to the CD-20 receptors on B-cells.

What is considered altered distribution?

- (1) Uptake in the lungs that is more intense than the heart on day one, or more intense than the liver on day 2 and 3.
- (2) Uptake in the kidneys more than the liver on day 3.
- (3) Uptake in the bowel that is fixed, and/or more than the liver
- (4) Uptake in the bone marrow > 25%

Trivia: Don't give to patients with platelets less than 100K

Most Common Side Effect? Thrombocytopenia and neutropenia (about 90% of cases).

Can you send them home post treatment? Dose to caretakers or persons near the patient is low, and they can be released to the general population after treatment. Although some things like sleeping apart, no kissing, etc... for about a week are still usually handed out.

Protocol: You need to first give rituximab to block the CD20 receptors on the circulating B cells and those in the spleen to optimize bio-distribution. Then you can give the In¹¹¹ labeled antibody to assess for altered bio-distribution. If you suspect altered distribution you should get delayed full body imaging at 90-120 hours. If altered you shouldn't treat. If ok, then blast'em.

Some High Yield Summary Charts

Tracer	Analog	Energy	Physical Half Life
Tc - 99m		"Low" - 140	6 hours
Iodine -123	Iodine	"Low" - 159	13 hours
Xenon - 133		"Low"-81	125 hours (<i>biologic t_{1/2} 30 seconds</i>)
Thallium - 201	Potassium	"Low"-135 (2%), 167 (8%), <i>use 71 ²⁰¹ Hg daughter x-rays</i>	73 hours
Indium -111		"Medium" - 173 (89%), 247 (94%)	67 hours
Gallium - 67	Iron	Multiple; 93 (40%), 184 (20%), 300 (20%), 393 (5%)	78 hours
Iodine -131	Iodine	"High" - 365	8 days
Fluorine -18	Sugar	"High"-511	110 mins

Treatment Radionuclides Half Life	
Strontium 89	50.5 DAYS (14 days in bone)
Samarium 153	46 Hours
Yttrium 90	64 Hours

Agent	Mechanism	Normal Distribution	Critical Organ
MDP	Phosphate analog - mechanism is " <i>Chemisorption</i> "	Bones, Kidneys	Renal
Free Xe-133	Not absorbed (inspired) - Can diffuse into fat	Airways	Trachea/Airways
MAA	Albumin - gets stuck in capillary (10-100 microns)	Lungs	Lungs
Sulfur Colloid	Phagocytized by RES	Liver, Spleen, Bone Marrow	Proximal Colon
HIDA	Bilirubin analog	Liver, GB, Bowel	Gallbladder Wall
WBC	Tagged Neutrophil - goes to RES, or area of active inflammation	Spleen, Liver, Bone Marrow	Spleen
RBC	Tagged RBC - circulated in blood	Blood Pool	Heart
MAG 3	Actively secreted	Kidneys, Bladder	Bladder
DMSA	Binds to sulhydryl group in proximal tubule	Kidneys	Kidney
Pertechnetate	Active Transport in the Thyroid	Stomach, Salivary Glands, Thyroid,	Stomach
Free Ga 67	Iron Analog, taken up by bacteria and WBC	Liver, Lacrimal and Salivary glands, Bones, Minimal Lungs	Distal Colon
FDG	Glucose Analog	Brain, Heart, Liver, Blood Pool	Bladder

Agent	Mechanism	Normal Distribution	Critical Organ
Sestamibi	Active transport	Salivary glands, GI tract, Heart, Kidneys, Thyroid parathyroid	Proximal Colon
Thallium 201	Potassium analog, Active transport (Na ⁺ /K pump)	Heart, Kidneys	Rental Cortex
Pentetreotide	Octreotide Analogue	Spleen, Liver, Kidneys, Bladder,	Spleen
MIBG	Analog of Norepinephrine. Enters neuroendocrine cells by an active uptake mechanism via the epinephrine transporter and is stored in the neurosecretory granules	Thyroid, Heart, Liver, Spleen, Salivary Gland	Spleen
HMPAO	Lipophilic, Crosses Blood Brain Barrier	Brain, Blood Pool	Bladder
DTPA (cistern)	Water Soluble	CSF, Kidney, Bladder	Spinal Cord
Iodine	Active Transport into the thyroid	Thyroid, Nasopharynx, Salivary Glands, Stomach, Bowel, Bladder	Thyroid



Exploiting the “Genius Neuron”

Have you ever heard someone in case conference take a case and lead with “It’s NOT this,” when clearly “this” is what the case was? It happens all the time. Often the first thing out of people’s mouths is actually the write answer, but many times you hear people say “it’s not” first. Ever wondered why?

There is this idea of a “Genius Neuron.” You have one neuron that is superior to the rest. This guy fires faster and is more reliable than his peers and because of this he is hated by them. He is the guy in the front row waving his hand shouting “I know the answer!” You know that guy, that guy is a notorious asshole. So, in your mind he shouts out the answer first, and then the rest of the neurons gang up on him and try and talk him out of it. So the end product is “It’s NOT this.”

For the purpose of taking cases in conference, this is why you should always lead with “this comes to mind,” instead of “it’s not.” Now, the practical piece of advice I want to give you is to trust your genius neuron. Serious, there is a lot of material on this test. But if you read this book, there will be enough knowledge to pass the test existing somewhere between your ears. You just have to trust that genius neuron.

How?? - Do it like this:

- (1) Read the entire question. Look at all the pictures.
- (2) Read ALL the answer choices. Never stop at A thinking that is the answer.
- (3) Look again at ALL the pictures - now that you see the choices.
- (4) Choose the first answer your mind tells you is correct - the one your genius neuron thinks it correct.
- (5) After you have finished the test, and you are re-reviewing your answers NEVER change the genius neuron’s answer except for two criteria. (A) You read the question wrong. (B) You are 100% sure that it is another choice, and you can give a reason why. Never change based on your gut feelings. Those secondary gut feelings are the stupid neurons trying to gang up on the smart one. Just like in the real world the stupid people significantly outnumber the smart ones.

I know this sounds silly, but I really believe in this. This is a real thing. I encourage you to try it with some practice questions.

Dealing with the Linked Question

It is a modern trend for multiple choice tests to have “linked” questions. You may remember that USMLE Step 3 had them, and it is rumored that the CORE Exam has them as well.

These are the questions that prompt you with “this is your final answer, you can’t change your answer.” When you see this STOP!

If you are 100% sure you are right, then go on. If you had it narrowed down to two choices, think about which one would be easier to write a follow up question about. This might seem obvious, but in the heat of the battle you might get too aggressive. Slow down and think twice on these.

The second point I want to make about these questions is finding some Zen if you miss it. There are a lot of questions on this test, it’s ok to miss some. You will still pass (probably). People like you have always studied for the A+, not the C-. So when you miss a question it makes you freak out because you think you blew it. You don’t need an A+ this time. You don’t need a B. You just need to pass so they don’t get any more money from you. Believe me they have taken enough from you already. I just want you to understand that you will miss questions and it’s ok. If the second part reveals that you dropped one, don’t let it phase you. Just do your best. The most important fight is always your next one.

Problem Solving Through MRI

Different programs have variable volume with MRI. Some of you will be excellent at it. Some of you will suck at it. An important skill to have is to understand how to problem solve with different sequences. The best way to do this is to have a list of T1 bright things, T2 bright things, dark things, and things that restrict diffusion.

T1 Bright	T2 Bright	T1 and T2 DARK	Restricts Diffusion
Fat	Fat	Flow Void	Stroke
Melanin (Melanoma)	Water	Fibrosis / Scar	Hypercellular Tumor
Blood (Subacute)	Blood (Extracellular Methemoglobin)	Metal	Epidermoid
Protein Rich Fluid	Most Tumors	Air	Abscess (Bacterial)
Calcification (Hyalinized)			Acute Demyelination
Slow Moving Blood			CJD
Laminar Necrosis			T2 Shine Through

Be able to move through sequences and problem solve.

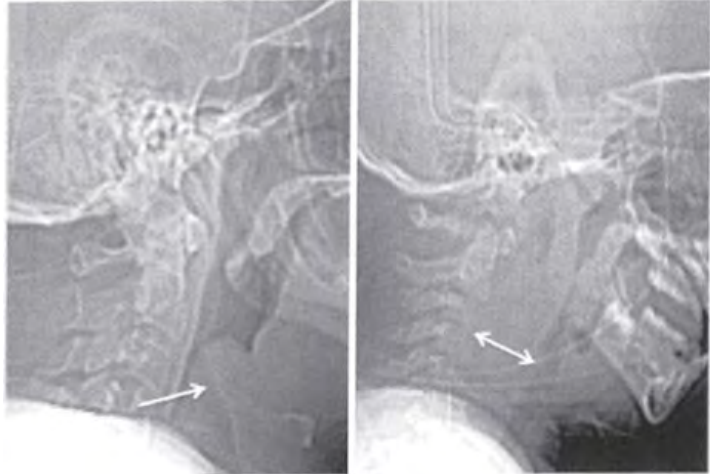
Think about a Lipoma for example. This will be T1 bright, T2 bright, and fat sat out. Another example might be something with layers in it. What can layer? Fat could layer, water could layer, blood could layer, pus could layer. Fat would be bright/ bright. Water would be dark on T1. Pus would be dark on T2. Blood could do different things depending on it's age. Fat would sat out. Pus may restrict diffusion (like a subdural empyema). You get the idea. Run through some scenarios in your mind. The key point is to know your differentials for this.

Section 1: Peds

Why are you showing me a lateral soft tissue neck x-ray?

This could be shown for two main reasons:

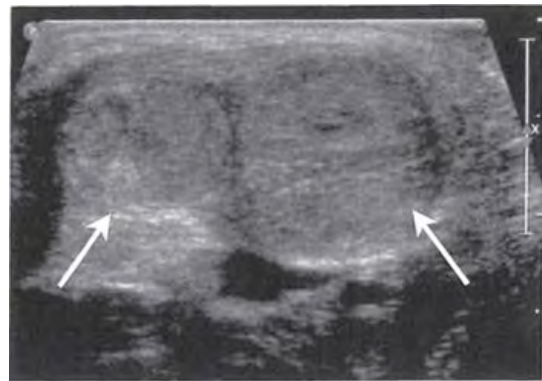
- (1) the thumb sign of epiglottitis, or
- (2) the super wide pre vertebral soft tissues for retropharyngeal abscess.



Epiglottitis - Thumb Sign Retropharyngeal Abscess

Why are you showing me an ultrasound of the neck? In peds this can only be a few things.

- **Fibromatosis Colli** is probably the most common thing shown (mass like fibrosis of the sternocleidomastoid muscle).
- Jugular vein thrombosis - think **Lemierre syndrome** (chest will have septic emboli). *Fusobacterium necrophorum*, is responsible for a majority of cases. •

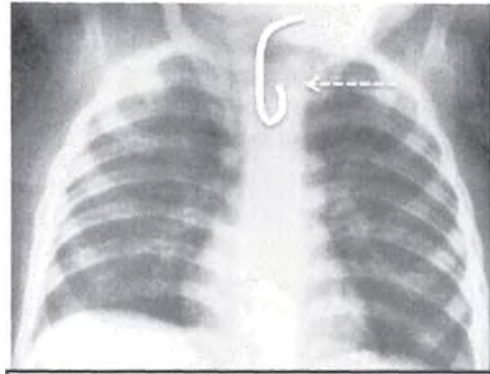


Fibromatosis Colli
(two heads of the sternocleidomastoid)

- Cysts
 - If midline think **Thyroglossal cyst**.
 - If lateral think **branchial cleft cyst**.

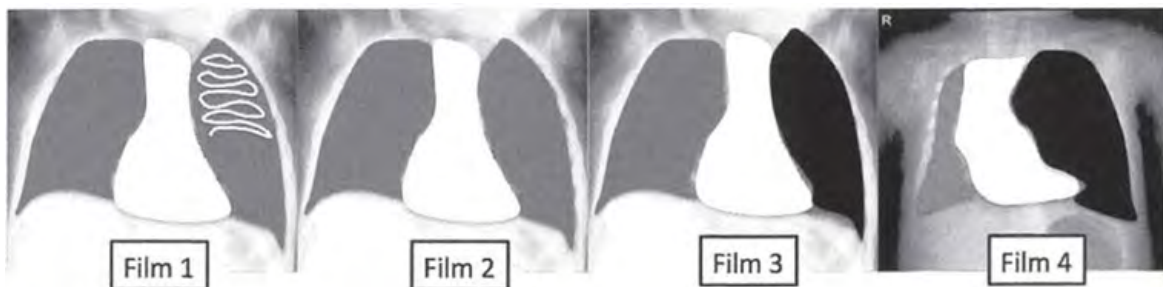
NG Tube Tricks: The presence of an NG tube (especially if not placed correctly) should alert you to some form of trickery.

- *The NG tube stops in the upper thoracic esophagus:* Think esophageal atresia (probably in the setting of VACTERL).
- *The NG tube curling into the chest - it's either (1) in the lung, or (2) it's in a congenital diaphragmatic hernia.* If I had to pick between the two (and it wasn't obvious), I'd say left side hernia, right side lung - just because those are the more common sides.



NG tube curls in the upper thoracic esophagus. First think Esophageal Atresia. Then think VACTERL

The Classic Congenital Lobar Emphysema Trick: They can show you a series of CXRs. The first one has an opacity in the lung (the affected lung is fluid filled). The next x-ray will show the opacity resolved. The following x-ray will show it getting more lucent, and more lucent. Until it's actually pushing the heart over. This is the classic way to show it in case conference, or case books



The Mandible: There are only a few things that a mandible will be shown for with regards to Peds. Think Caffey's first - especially if the picture looks blurry and old (there hasn't been a case of this in 50 years). If it's osteonecrosis think about O.I. on bisphosphonates. If it's a dwarf case, think wide angled mandible with Pycnodysostosis. A "floating tooth" could be EG.

The School Aged CXR: *Things to look for:*

- **Big Heart** - Probably showing you a sickle cell case. Look for bone infarcts in shoulders.
- **Lucent Lung** - Think foreign Body (air trapping). Remember you put the affected side down (if it remains lucent- that confirms it).

The Abdominal Plain Film - on a newborn - Problem Solving Bubbles:

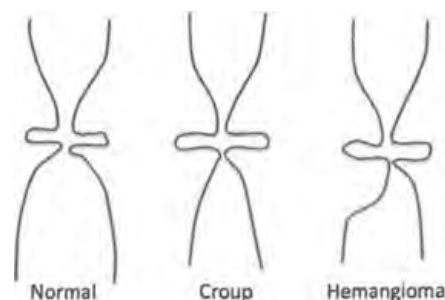
Pattern	Path	Next Step
Single Bubble:	In a newborn this is Gastric (antral or pyloric atresia). In an older child think gastric volvulus	
Double Bubble	Duodenal Atresia	
Triple Bubble	Jejunal Atresia	
Single Bubble + Distal Gas + "Bilious Vomiting	Concern for Mid Gut Volvulus	Next Step = Upper GI
Multiple Dilated Loops	Concern for lower obstruction	Next Step = Contrast Enema

Heterotaxia: This can be inferred or asked several ways.

Heterotaxia Syndromes	
Right Sided	Left Sided
Two Fissures in Left Lung	One Fissure in Right Lung
Asplenia	Polysplenia
Increased Cardiac Malformations	Less Cardiac Malformations
Reversed Aorta/IVC	Azygous Continuation of the IVC

This vs That:

The “steeple sign” of croup affects both subglottic shoulders, a subglottic hemangioma is unilateral.

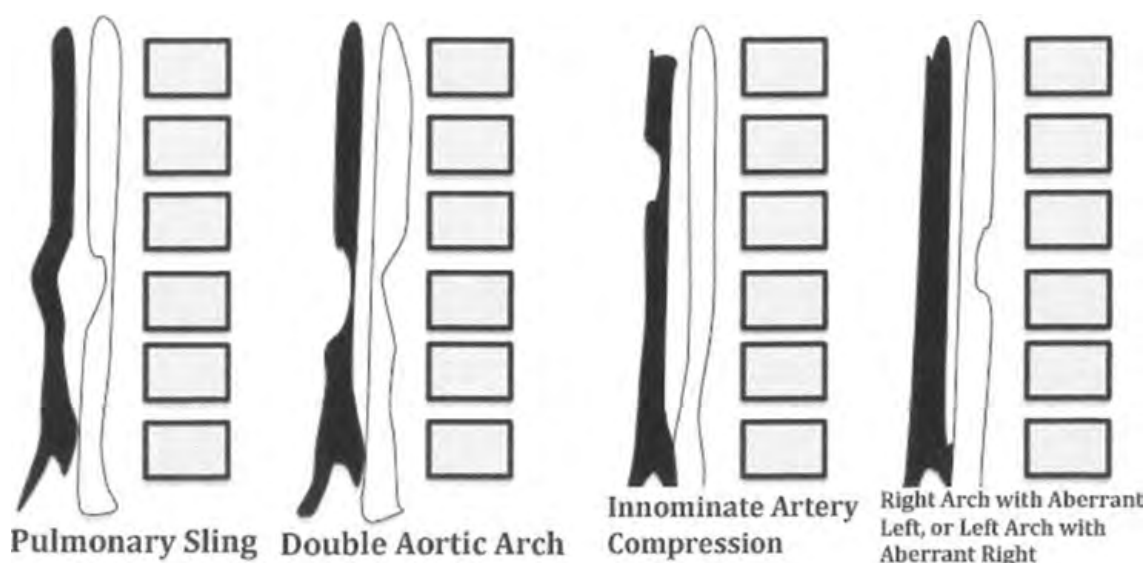


This vs That: *Neonatal pneumonia and RDS* can look similar (both with granular opacities). RDS should have low lung volumes (regular pneumonia will have increases). RDS is gonna be premature (if they give you that history). Lastly, pneumonia will often have a pleural effusion - which RDS will not.

This vs That: *Cystic Fibrosis vs Primary Ciliary Dyskinesia*

CF	PCD
Abnormal Mucus - Cilia can't clear it	Normal Mucus - Cilia don't work
Bronchiectasis (lower lobes)	Bronchiectasis (upper lobes)
Normal sperm, obliterated vas deferens	Normal vas deferens, sperm cannot swim normally

This vs That: *Vascular Impressions:*

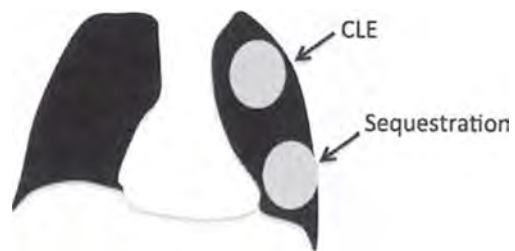


This vs That: *Intralobar vs Extralobar Sequestration*

Intralobar	Extralobar
Has pleural covering	No pleural covering
More Common	Less Common
Presents later with recurrent infection	Presents early with other bad congenital things (heart, etc...)

This vs That - Location:

- Intralobar Sequestration = Left Lower Lobe
- Congenital Lobar Emphysema (CLE) = Left Upper Lobe
- CCAM = No Lobar Preference



This vs That: Duodenal Atresia vs Jejunal Atresia:

Duodenal Atresia	Jejunal Atresia
Double Bubble	Triple Bubble
Failure to Canalize (often isolated atresia)	Vascular Insult * More likely associated with other atresias
Associated with Downs	

Baby Liver - This vs That:

Age 0-3		
Hemangioendothelioma	Endothelial Growth Factor Elevated	High flow heart failure, big heart on CXR.
Hepatoblastoma	AFP Elevated	Associated with Wilms, Associated with Prematurity Can cause precocious puberty
Mesenchymal Hamartoma	AFP Negative	It's really cystic

Peds Cystic Renal Mass



Multi Cystic Dysplastic Kidney

- *Neonate, No Renal Function*

AR-PCKD

- *Enlarged, Bright, Microcystic*

Multilocular Cystic Nephroma

- *Young Boy, "Herniates into Renal Pelvis"*

Cystic Wilms

This vs That:

Neuroblastoma	Wilms
Age: usually less than 2 (can occur in utero)	Age: Usually around are 4 (never before 2 months)
Calcifies 90%	Calcifies Rarely (<10%)
Encases Vessels (doesn't invade)	Invades Vessels (doesn't encase)
Poorly Marginated	Well Circumscribed
Mets to Bones	Doesn't usually met to bones (unless clear cell wilms variant).

This vs That:

Neuroblastoma	Adrenal Hemorrhage
Anechoic and Avascular	Echogenic and Vascular
Low of T2	Hight on T2
Will grow on followup	Should shrink on followup

When I Say This.....You Say That.....

- When I say “Subglottic Hemangioma,” You Say PHACES Syndrome
- When I say “PHACES Syndrome,” You say Cutaneous Hemangioma
- When I say “Ropy Appearance,” You say Meconium Aspiration
- When I say “Post Term Delivery,” You Say Meconium Aspiration
- When I say “Fluid in the Fissures,” You say Transient Tachypnea
- When I say “History of c-section”, You say Transient Tachypnea
- When I say “Maternal sedation”, You say Transient Tachypnea
- When I say “Granular Opacities + Premature”, You say RDS
- When I say “Granular Opacities + Term + High Lung Volume,” You say Pneumonia
- When I say “Granular Opacities + Term + Low Lung Volume,” You say B-Hemolytic Strep
- When I say “Band Like Opacities”, You say Chronic Lung Disease (BPD)
- When I say “Linear Lucencies” , You say Pulmonary Interstitial Emphysema
- When I say “Pulmonary Hypoplasia,” You say diaphragmatic hernia
- When I say “Lung Cysts and Nodules,” You Say LCH or Papillomatosis
- When I say “Lower lobe bronchiectasis,” You Say Primary Ciliary Dyskinesia
- When I say “Upper lobe bronchiectasis,” You Say CF
- When I say “Posterior mediastinal mass (under 2),” You Say Neuroblastoma
- When I say “No air in the stomach”, You say Esophageal Atresia
- When I say “Excessive air in the stomach”, You say “H” Type TE fistula
- When I say “Anterior Esophageal Impression,” You say pulmonary sling
- When I say “Pulmonary Sling,” You say tracheal stenosis.
- When I say “Single Bubble,” You say Gastric (antral or pyloric) atresia
- When I say “Double Bubble,” You say duodenal atresia
- When I say “Duodenal Atresia”, You say Downs
- When I say “Single Bubble with Distal Gas,” You say maybe Mid Gut Volvulus
- When I say “Non-bilious vomiting”, You say Hypertrophic Pyloric Stenosis
- When I say “Paradoxial aciduria” You say Hypertrophic Pyloric Stenosis
- When I say “Bilious vomiting - in an infant”, You say Mid Gut Volvulus
- When I say “Corkscrew Duodenum” You say Mid Gut Volvulus
- When I say “Reversed SMA and SMV” You say Malrotation
- When I say “Absent Gallbladder” You say biliary atresia
- When I say “Triangle Cord Sign” You say biliary atresia
- When I say “Asplenia” , You say “cyanotic heart disease”

- When I say “Infarcted Spleen,” You say Sick Cell
- When I say “Gall Stones,” You say Sick Cell
- When I say “Short Microcolon,” You say Colonic Atresia
- When I say “Long Microcolon,” You say Meconium ileus or distal ileal atresia
- When I say “Saw tooth colon,” You say Hirschsprung
- When I say “Calcified mass in the mid abdomen of a newborn”, you say Meconium Peritonitis
- When I say “Meconium ileus equivalent,” you say Distal Intestinal Obstruction Syndrome (CF).
- When I say “Abrupt caliber change of the aorta below the celiac axis” , You say Hepatic Hemangioendothelioma.
- When I say “Cystic mass in the liver of a newborn,” you say Mesenchymal Hamartoma
- When I say “Elevated AFP, with mass in the liver of a newborn,” you say Hepatoblastoma
- When I say “Common Bile Duct measures more than 10mm”, You say Choledochal Cyst
- When I say “Lipomatous pseudohypertrophy of the pancreas,” You say CF
- When I say “Unilateral Renal Agenesis” You say unicornuate uterus
- When I say “Neonatal Renal Vein Thrombosis,” You say maternal diabetes
- When I say “Neonatal Renal Artery Thrombosis,” You say Misplaced Umbilical Artery Catheter
- When I say “Hydro on Fetal MRI,” You say Posterior Urethral Valve
- When I say “Urachus,” You say bladder Adenocarcinoma
- When I say “Nephroblastomatosis with Necrosis,” you say Wilms
- When I say “Solid Renal Tumor of Infancy,” you say Mesoblastic Nephroma
- When I say “Solid Renal Tumor of Childhood,” you say Wilms
- When I say “Midline pelvic mass, in a female,” you say Hydrometrocolpos
- When I say “Right sided varicocele,” you say abdominal pathology
- When I say “Blue Dot Sign,” you say Torsion of the Testicular Appendages
- When I say “Hand or Foot Pain / Swelling in an Infant”, You say - sickle cell with hand foot syndrome.
- When I say Extratesticular scrotal mass, you say embryonal rhabdomyosarcoma
- When I say “Narrowing of the interpedicular distance,” you say Achondroplasia
- When I say “Platyspondyly (flat vertebral bodies),” you say Thanatophoric

High Yield Trivia:

- Pulmonary Interstitial Emphysema (PIE) - put the bad side down
- Bronchial Foreign Body - put the lucency side down (if it stays that way, it's positive)
- Papillomatosis has a small (2%) risk of squamous cell CA
- Pulmonary sling is the only variant that goes between the esophagus and the trachea. This is associated with trachea stenosis.
- Thymic Rebound - Seen after stress (chemotherapy) - Can be PET-Avid
- Lymphoma - Most common mediastinal mass in child (over 10)
- Anterior Mediastinal Mass with Calcification - Either treated lymphoma, or Thymic Lesion (lymphoma doesn't calcify unless treated).
- Neuroblastoma is the most common posterior mediastinal mass in child under 2 (primary thoracic does better than abd).
- Hypertrophic Pyloric Stenosis - NOT at birth, NOT after 3 months (3 weeks to 3 months is easiest for me to remember)
- Criteria for HPS - 4mm and 14mm (4mm single wall, 14mm length).
- Annular Pancreas presents as duodenal obstruction in children and pancreatitis in adults.
- Most common cause of bowel obstruction in child over 4 = Appendicitis
- Intussusception - 3months to 3 years is ok, earlier or younger think lead point
- Gastroschisis is ALWAYS on the right side
- Omphalocele has associated anomalies (gastroschisis does not).
- Physiologic Gut Hernia normal at 6-8 weeks
- AFP is elevated with Hepatoblastoma
- Endothelial growth factor is elevated with Hemangioendothelioma
- Most Common cause of pancreatitis in a kid = Trauma (seatbelt)
- Weigert Meyer Rule - Duplicated ureter on top inserts inferior and medial
- Most common tumor of the fetus or infant - Sacrococcygeal Teratoma
- Most common cause of idiopathic scrotal edema - HSP
- Most common cause of acute scrotal pain age 7-14 - Torsion of Testicular Appendages
- Bell Clapper Deformity is the etiology for testicular torsion.
- SCFE is a Salter Harris Type 1
- Physiologic Periostitis of the Newborn doesn't occur in a newborn - seen around 3 months
- Acetabular Angle should be < 30 , and Alpha angle should be more than 60.

Section 2: GI

Classic Gamesmanship: They could show you a dilated esophagus on CT or barium. Then they show you ground glass in the lung bases (and sub-pleural sparing if you are lucky). This answer is scleroderma with NSIP.

Benign Liver Masses					
	Ultrasound	CT	MR	Trivia	
Hemangioma	Hyperechoic	Peripheral Nodular Discontinuous Enhancement	T2 Bright	Rare in Cirrhotics	
FNH	Spoke Wheel	Homogenous Arterial Enhancement	“Stealth Lesion - Iso on T1 and T2”	Central Scar	Bright on Delayed Eovist (Gd-EOB-DTPA)
Hepatic Adenoma	Variable	Variable	Fat Containing on In/Out Phase	OCP use, Glycogen Storage Disease	Can explode and bleed
Hepatic Angiomyolipoma	Hyperechoic	Gross Fat	T1/T2 Bright	Unlike renal AML, 50% don't have fat	Tuberous Sclerosis

Location Location:

- **H Pylori** Gastritis — Usually in **Antrum**
- **Zollinger-Ellison** - Ulcerations in the stomach (**jejunal ulcer** is the buzzword). Duodenal bulb is actually the most common location for ulcers in ZE.
- **Crohns** - Uncommon in the stomach, but when it is, it likes the **antrum**
- **Menetrier's** - Usually in the **Fundus** (*classically spares the antrum*)
- **Lymphoma** - “**Crosses the Pylorus**” - classically described as doing so, although in reality adenocarcinoma does it more.

Infections'.

- **Giardia** - Duodenum
- **Strongyloides** - Duodenum
- **TB** - Terminal Ileum
- **Yersinia** - Terminal Ileum

This vs That:

- Herpes Esophagitis = Multiple Small Ulcers
- CMV and AIDS = Solitary Large Ulcer

This vs That - Esophageal Cancer:

- Squamous Cell = Black Guy who drinks and smokes - mid esophagus
- Adenocarcinoma = White Guy with reflux (history of PPIs), - lower esophagus

This vs That - Uphill vs Downhill Varices

Uphill Varices	Downhill Varices
Caused by Portal Hypertension	Caused by SVC obstruction (catheter related, or tumor related)
Confined to Bottom Half of Esophagus	Confined to Top Half of Esophagus

This vs That - Traction vs Pulsion Diverticulum

Traction	Pulsion
Triangular	Round
Will Empty	Will NOT Empty (contain no muscle in their walls)

This vs That - Esophageal Hernias

- Sliding - GE Junction Above the Diaphragm
- Rolling - GE Junction Below the Diaphragm

This vs That - Carney's Triad vs Carney's Complex

Carney's Triad	Carney's Complex
Extra-Adrenal Pheochromocytoma,	Cardiac Myxoma * (C for Complex)
GIST	Skin Stuff
Pulmonary Chordoma (hamartoma)	Endocrine Stuff

This vs That - Benign vs Malignant Ulcers (on Barium)

Malignant	Benign
Width > Depth	Depth > Width
Located within Lumen	Project behind the expected lumen
Nodular, Irregular Edges	Sharp Contour
Folds adjacent to ulcer	Folds radiate to ulcer
Aunt Minnie: Carmen Meniscus Sign	Aunt Minnie: Hampton's Line

This vs That - Inguinal Hernia

Direct	Indirect
Less common	More Common
Medial to inferior Epigastric	Lateral to inferior epigastric
Defect in Hesselbach triangle	Failure of processus vaginalis to close
NOT covered by internal spermatic fascia	Covered by internal spermatic fascia

This vs That - Crohns and UC

Crohns	UC
Slightly less common in the USA	Slightly more common in the USA
Discontinuous "Skips"	Continuous
Terminal Ileum - <i>String Sign</i>	Rectum
Ileocecal Valve "Stenosed"	Ileocecal Valve "Open"
Mesenteric Fat Increased " <i>creepingfat</i> "	Perirectal fat Increased
Lymph nodes are usually enlarged	Lymph nodes are NOT usually enlarged
Makes Fistula	Doesn't Usually Make Fistula

This vs That - Volvulus

Sigmoid	Cecal
Old Person (Constipated)	Younger Person (mass, prior surgery, or 3rd Trimester Pregnancy)
Points to the RUQ	Points to the LUQ

This vs That -Liver Nodules

Regenerative	Dysplastic	HCC
Contains Iron	Contains Fat, Glycoprotein	
T1 Dark, T2 Dark	T1 Bright, T2 Dark	T2 Bright
Does NOT Enhance	Usually Does NOT Enhance	Does Enhance

This vs That -Central Scars

FNH	FL HCC
T2 Bright	T2 Dark (usually)
Enhances on Delays	Does NOT enhance
Mass is Sulfur Colloid Avid (sometimes)	Mass is Gallium Avid

This vs That -Hepatic Adenoma vs FNH

Hepatic Adenoma	FNH
Usually > 8cm	Usually < 8cm
No Bile Ducts	Normal Bile Ducts
No Kupffer Cells	Normal Kupffer Cells
Sulfur Colloid Cold	Sulfur Colloid Hot (sometimes)

This vs That -HCC vs Fibrolamellar HCC

HCC	FL HCC
Cirrhosis	No Cirrhosis
Older (50s-60s)	Young (30s)
Rarely Calcifies	Calcifies Sometimes
Elevated AFP	Normal AFP

This vs That -Hemochromatosis - Primary vs Secondary

Primary	Secondary
Genetic - increased absorption	Acquired - chronic illness, and multiple transfusions
Liver, Pancreas	Liver, Spleen
Heart, Thyroid, Pituitary	

When I Say This.....You Say That.....

- When I say “narrowed B Ring,” You say Schatzki
- When I say “esophageal concentric rings,” You say Eosinophilic Esophagitis
- When I say “shaggy” or “plaque like” esophagus, You say Candidiasis
- When I say “looks like Candida, but an asymptomatic old lady,” you say Glycogen Acanthosis
- When I say “reticular mucosal pattern,” you say Barretts
- When I say “high stricture with an associated hiatal hernia,” you say Barretts
- When I say “abrupt shoulders,” you say cancer
- When I say “Killian Dehiscence,” you say Zenker Diverticulum
- When I say “transient, fine transverse folds across the esophagus,” you say Feline Esophagus.
- When I say “bird’s beak,” you say Achalasia
- When I say “solitary esophageal ulcer,” you say CMV or AIDS
- When I say “ulcers at the level of the arch or distal esophagus,” you say Medication induced
- When I say “Breast Cancer + Bowel Hamartomas,” you say Cowdens
- When I say “Desmoid Tumors + Bowel Polyps,” you say Gardners
- When I say “Brain Tumors + Bowel Polyps,” you say Turcots
- When I say “enlarged left supraclavicular node,” you say Virchow Node (GI Cancer)
- When I say “crosses the pylorus,” you say Gastric Lymphoma
- When I say “isolated gastric varices,” you say splenic vein thrombus
- When I say “multiple gastric ulcers,” you say Chronic Aspirin Therapy.
- When I say “multiple duodenal (or jejunal) ulcers,” you say Zollinger-Ellsion
- When I say “pancreatitis after Billroth 2,” you say Afferent Loop Syndrome
- When I say “Weight gain years after Roux-en-Y,” you say Gastro-Gastro Fistula
- When I say “Clover Leaf Sign - Duodenum,” you say healed peptic ulcer.
- When I say “Sand Like Nodules in the Jejunum,” you say Whipples
- When I say “Sand Like Nodules in the Jejunum + CD4 <100,” you say MAI
- When I say “Ribbon-like bowel,” you say Graft vs Host
- When I say “Ribbon like Jejunum,” you say Long Standing Celiac
- When I say “Moulage Pattern,” you say Celiac
- When I say “Fold Reversal - of jejunum and ileum,” you say Celiac
- When I say “Cavitary (low density) Lymph nodes,” you say Celiac

- When I say “hide bound” or “Stack or coins,” you say Scleroderma
- When I say “Megaduodenum,” you say Scleroderma
- When I say “Duodenal obstruction, with recent weight loss,” you say SMA Syndrome
- When I say “Coned shaped cecum,” you say Amebiasis
- When I say “Lead Pipe,” you say Ulcerative Colitis
- When I say “String Sign,” you say Crohns
- When I say “Massive circumferential thickening, without obstruction,” you say Lymphoma
- When I say “Multiple small bowel target signs,” you say Melanoma
- When I say “Obstructing Old Lady Hernia,” you say Femoral Hernia
- When I say “sac of bowel,” you say Paraduodenal hernia.
- When I say “scalloped appearance of the liver,” you say Pseudomyxoma Peritonei
- When I say “HCC without cirrhosis,” you say Hepatitis B
- When I say “Capsular retraction,” you say Cholangiocarcinoma
- When I say “Periportal hypoechoic infiltration + AIDS,” you say Kaposi’s
- When I say “sparing of the caudate lobe,” you say Budd Chiari
- When I say “large T2 bright nodes + Budd Chiari,” you say Hyperplastic nodules
- When I say “liver high signal in phase, low signal out phase,” you say fatty liver
- When I say “liver low signal in phase, and high signal out phase,” you say hemochromatosis
- When I say “multifocal intrahepatic and extrahepatic stricture,” you say PSC
- When I say “multifocal intrahepatic and extrahepatic strictures + papillary stenosis,” you say AIDS Cholangiopathy.
- When I say “bile ducts full of stones,” you say Recurrent Pyogenic Cholangitis
- When I say “Gallbladder Comet Tail Artifact,” you say Adenomyomatosis
- When I say “lipomatous pseudohypertrophy of the pancreas,” you say CF
- When I say “sausage shaped pancreas,” you say autoimmune pancreatitis
- When I say “autoimmune pancreatitis,” you say IgG4
- When I say “IgG4” you say RP Fibrosis, Sclerosing Cholangitis, Fibrosing Medianstinitis, Inflammatory Pseudotumor
- When I say “Wide duodenal sweep,” you say Pancreatic Cancer

High Yield Trivia

- Most Common benign mucosal lesion of the esophagus = Papilloma
- Esophageal Webs have increased risk for cancer, and Plummer-Vinson Syndrome (anemia + web)
- Dysphagia Lusoria is from compression by a right subclavian artery (most patients with aberrant rights don't have symptoms).
- Achalasia has an increased risk of squamous cell cancer (20 years later).
- Most common mesenchymal tumor of the GI tract = GIST
- Most common location for GIST = Stomach
- Krukenberg Tumor = Stomach (GI) met to the ovary
- Menetrier's : involves fundus and spares the antrum
- The stomach is the most common location for sarcoid (in the GI tract)
- Gastric Remnants have an increased risk of cancer years after Billroth
- Most common internal hernia, Left sided paraduodenal
- Most common site of peritoneal carcinomatosis = retrovesical space
- An injury to the bare area of the liver can cause a retroperitoneal bleed
- Primary Sclerosing Cholangitis associated with Ulcerative Colitis
- Extrahepatic ducts are normal with Primary Biliary Cirrhosis
- Antimitochondrial Antibodies - positive with primary biliary cirrhosis
- Mirizzi Syndrome - the stone in the cystic duct obstructs the CBD.
- Mirizzi has a 5x increased risk of GB cancer.
- Dorsal pancreatic agenesis - associated with diabetes and polysplenia
- Hereditary and Tropical Pancreatitis - early age of onset, increased risk of cancer
- When I say "Grandmother Pancreatic Cyst" you say Serous Cystadenoma
- When I say "Mother Pancreatic Cyst" you say Mucinous
- When I say "Daughter Pancreatic Cyst," you say Solid Pseudopapillary
- Felty's Syndrome - Big Spleen, RA, and Neutropenia
- Splenic Artery Aneurysm - more common in women, and more likely to rupture in pregnant women.
- Insulinoma is the most common islet cell tumor
- Gastrinoma is the most common islet cell tumor with MEN
- Ulcerative Colitis has an increased risk of colon cancer (if it involves colon past the splenic flexure). UC involving the rectum only does not increase risk of CA.

Section 3: GU

Gamesmanship: Showing persistent nephrograms - either by plain film or CT is the classic trick for ATN - usually contrast induced nephropathy

Gamesmanship: If you are show a unilateral renal agenesis case, remember the associations with absent ipsilateral epididymis, absent vas deferens, and ipsilateral seminal vesicle cyst in a man. For a woman think about mullarian anomalies (unicornuate uterus).

Renal Cancer Syndromes

Subtype	Syndrome / Association
Clear Cell	Von Hippel-Lindau
Papillary	Hereditary papillary renal carcinoma
Chromophobe	Birt Hogg Dube
Medullary	Sickle Cell Trait

Renal Cysts Syndromes:

ADPKD	Cysts in Liver	Kidneys are BIG
VHL	Cysts in Pancreas	
Acquired (Uremic)		Kidneys are small

Gamesmanship: If they wanted to ask Oncocytoma they can show it 3 ways: (1) On CT Solid Mass with Central Scar, (2) On Ultrasound “spoke wheel” vascular pattern, (3) on PET CT it will be hotter than surrounding renal cortex.

Gamesmanship: RCC is typically colder than surrounding renal parenchyma on PET, whereas oncocytoma is typically hotter.

Gamesmanship: IVPs haven’t been used since the 1970s. If you see one there are a few tricks. The most common is the medial deviation of the ureters (retroperitoneal fibrosis), or the lateral deviation of the ureters (Psoas Hypertrophy, or lymph nodes).

When I Say This..... You Say That

- When I say “bladder stones,” you say neurogenic bladder
- When I say “pine cone appearance,” you say neurogenic bladder
- When I say “urethra cancer,” you say squamous cell CA
- When I say “urethra cancer - prostatic portion,” you say transitional cell CA
- When I say “urethra cancer - in a diverticulum,” you say adenocarcinoma
- When I say “vas deferens calcifications,” you say diabetes
- When I say “calcifications in a fatty renal mass,” you say RCC
- When I say “protrude into the renal pelvis,” you say Multilocular cystic nephroma
- When I say “no functional renal tissue,” you say Multicystic Dysplastic Kidney
- When I say “Multicystic Dysplastic Kidney,” you say contralateral renal issues (50%)
- When I say “Emphysematous Pyelonephritis,” you say diabetic
- When I say “Xanthogranulomatous Pyelonephritis,” you say staghorn stone
- When I say “Papillary Necrosis,” you say diabetes
- When I say “shrunk calcified kidney,” you say TB
- When I say “big bright kidney with decreased renal function,” you say HIV
- When I say “history of lithotripsy,” you say Pyelo Kidney
- When I say “cortical rim sign,” you say subacute renal infarct
- When I say “history of renal biopsy,” you say AVF
- When I say “reversed diastolic flow,” you say renal vein thrombosis
- When I say “sickle cell trait,” you say medullary RCC
- When I say “Young Adult, Renal Mass, + Severe HTN,” you say Juxtaglomerular Cell Tumor
- When I say “squamous cell bladder CA,” you say Schistosomiasis
- When I say “entire bladder calcified,” you say Schistosomiasis
- When I say “urachus,” you say adenocarcinoma of the bladder
- When I say “long stricture in urethra,” you say Gonococcal
- When I say “short stricture in urethra,” you say Straddle Injury

High Yield Trivia

- Calcifications in a renal CA - are associated with an improved survival
- RCC bone mets are “always” lytic
- There is an increased risk of malignancy with dialysis
- Horseshoe kidneys are more susceptible to trauma
- Most common location for TCC is the bladder
- Second most common location for TCC is the upper urinary tract
- Upper Tract TCC is more commonly multifocal (12%) - as opposed to bladder (4%).
- Weigert Meyer Rule - Upper Pole inserts medial and inferior
- Ectopic Ureters are associated with incontinence in women (not men)
- Leukoplakia is pre-malignant; Malakoplakia is not pre-malignant
- Extraperitoneal bladder rupture is more common, and managed medically
- Intraperitoneal bladder rupture is less common, and managed surgically
- Indinavir stones are the only ones not seen on CT.
- Uric Acid stones are not seen on plain film.

Section 4: Reproductive

This vs That - *Bicornuate vs Septate Uterus*

You distinguish the two by the apex of the fundal contour:

- * *Apex of Fundal Contour > 5mm Above Tubal Ostia = Septate*
- * *Apex of Fundal Contour < 5 mm Above Tubal Ostia = Bicornuate*
- * *Other important trivia is; Septate has established increased 1st trimester loss, bicornuates have a lot less problems (maybe no increased risk - depends on who you ask).*

This vs That: *Gartner Duct Cyst vs Bartholin Cyst*

- The Gartner duct cyst is above the pubic symphysis (Bartholin is below it).

This vs That: *Central Gland Prostate CA vs BPH*

- Prostate CA is usually in the peripheral zone (not the central zone). When it is in the central zone it's T2 "smudgy" or charcoal.
- BPH nodules are usually in the central zone, and they have a sharp border. You can "draw a line around them with a pencil."

This vs That: *Symmetrical vs Asymmetrical - IUGR*

Symmetrical is a "placenta problem." There is sparing of the head. It's normal until the 3rd trimester. Causes include Maternal Hypertension, Severe Malnutrition, and Ehler-Danlos.

Asymmetrical is a "baby problem." The head is NOT spared. It's seen early, including the first trimester. Causes include TORCHS, Fetal EtOH, and Chromosomal Abnormalities.

Gamesmanship - The combination of an ovarian mass and a thickened endometrium should make you think Granulosa Cell Tumors (estrogen making).

Gamesmanship - Seeing fluid in the endometrial canal of a postmenopausal woman should make you think the cervix is obstructed (either by cancer, or more commonly stenosis).

Gamesmanship: A met to the vagina in the anterior wall upper 1/3 is "always" (90%) upper genital tract. A met to the vagina in the posterior wall lower 1/3 is "always" (90%) from the GI tract.

Gamesmanship: Peritoneal inclusion cysts occur after abdominal surgery (from adhesions). If they mention in the question stem “history of abdominal surgery” , and it’s a GYN case have that on your radar.

Gamesmanship: If “hyperemesis” is in the the question stem, think about things that give you an elevated B-hCG - like moles and multiple pregnancy (twins).

Gamesmanship: If they show you a varicocele, regardless of what side it’s on (right being more suspicious than left), and it’s a “next step” type deal you probably should look for the abdominal cancer.

When I Say This.....You Say That.....

- When I say “Unicornuate Uterus,” you say Look at the kidneys
- When I say “T-Shaped Uterus,” you say DES related or Vaginal Clear Cell CA
- When I say “Marked enlargement of the uterus,” you say Adenomyosis
- When I say “Adenomyosis,” you say thickening of the junctional zone (> 12mm)
- When I say “Wolffian duct remnant,” you say Gartner Duct Cyst
- When I say “Theca Lutein Cysts,” you say moles and multiple gestations
- When I say “Theca Lutein Cysts + Pleural Effusions,” you say - Hyperstimulation Syndrome (patient on fertility meds).
- When I say “Low level internal echoes,” you say Endometrioma
- When I say “T2 Shortening,” you say - Endometrioma - “Shading Sign”
- When I say “Fishnet appearance,” you say Hemorrhagic Cyst
- When I say “Ovarian Fibroma + Pleural Effusion,” you say Meigs Syndrome
- When I say “Snow Storm Uterus,” you say Complete Mole - 1st Trimester
- When I say “Serum P-hCG levels that rise in the 8 to 10 weeks following evacuation of molar pregnancy,” you say Choriocarcinoma
- When I say “midline cystic structure near the back of the bladder of a man,” you say Prostatic Utricle
- When I say “lateral cystic structure near the back of the bladder of a man,” you say Seminal Vesicle Cyst
- When I say “isolated orchitis,” you say mumps
- When I say “onion skin appearance,” you say epidermoid cyst
- When I say “multiple hypoechoic masses in the testicle,” you say lymphoma
- When I say “cystic elements and macro-calcifications in the testicle,” you say Mixed Germ Cell Tumor
- When I say “homogenous and microcalcifications,” you say seminoma

- When I say “gynecomastia + testicular tumor,” you say Sertoli Leydig
- When I say “fetal macrosomia,” you say Maternal Diabetes
- When I say “one artery adjacent to the bladder,” you say two vessel cord
- When I say “painless vaginal bleeding in the third trimester,” you say placenta previa
- When I say “mom doing cocaine,” you say placenta abruption
- When I say “thinning of the myometrium - with turbulent doppler,” you say placenta creta
- When I say “mass near the cord insertion, with flow pulsating at the fetal heart rate,” you say placenta chorioangioma
- When I say “Cystic mass in the posterior neck -antenatal period,” you say cystic hygroma.
- When I say “Pleural effusions, and Ascites on prenatal US,” you say hydrops.
- When I say “Massively enlarged bilateral kidneys,” you say ARPKD
- When I say “Twin peak sign,” you say dichorionic diamniotic

High Yield Trivia

- Endometrial tissue in a rudimentary horn (even one that does NOT communicate) increases the risk of miscarriage.
- Arcuate Uterus does NOT have an increased risk of infertility (it’s a normal variant)
- Fibroids with higher T2 signal respond better to UAE
- Hyaline Fibroid Degeneration is the most common subtype
- Adenomyosis - favors the posterior wall, spares the cervix
- Hereditary Non-Polyposis Colon Cancer (NHPCC) - have a 30-50x increased risk of endometrial cancer
- Tamoxifen increases the risk of endometrial cancer, and endometrial polyps
- Cervical Cancer that has parametrial involvement (2B) - is treated with chemo/radiation. Cervical Cancer without parametrial involvement (2A) - is treated with surgery
- Vaginal cancer in adults is usually squamous cell
- Vaginal Rhabdomyosarcoma occurs in children / teenagers
- Premenopausal ovaries can be hot on PET (depending on the phase of cycle). Post menopausal ovaries should Never be hot on PET.
- Transformation subtypes: Endometrioma = Clear Cell, Dermoid = Squamous
- Post Partum fever can be from ovarian vein thrombophlebitis
- Fractured penis = rupture of the corpus cavernosum and the surrounding tunica albuginea.

- Prostate Cancer is most commonly in the peripheral zone, - ADC dark
- BPH nodules are in the central zone
- Hypospadias is the most common association with prostatic utricle
- Seminal Vesicle cysts are associated with renal agenesis, and ectopic ureters
- Cryptorchidism increases the risk of cancer (in both testicles), and is not reduced by orchiopexy
- Immunosuppressed patients can get testicular lymphoma -hiding behind blood testes barrier
- Most common cause of correctable infertility in a man is a varicocele.
- Undescended testicles are more common in premature kids.
- Membranes disrupted before 10 weeks, increased risk for amniotic bands
- The earliest visualization of the embryo is the “double bleb sign”
- Hematoma greater than 2/3 the circumference of the chorion has a 2x increased risk of abortion.
- Biparietal Diameter - Recorded at the level of the thalamus from the outermost edge of the near skull to the inner table of the far skull.
- Abdominal Circumference - does not include the subcutaneous soft tissues
- Abdominal Circumference is recorded at the the level of the junction of the umbilical vein and left portal vein
- Femur Length does NOT include the epiphysis
- Umbilical Artery Systolic / Diastolic Ratio should NOT exceed 3 at 34 weeks - makes you think pre-eclampsia and IUGR
- A full bladder can mimic a placenta previa
- Nuchal lucency is measured between 9-12 weeks, and should be < 3mm. More than 3mm is associated with downs.
- Lemon sign will disappear after 24 weeks
- Aqueductal Stenosis is the most common cause of non-communicating hydrocephalus in a neonate

Section 5: Chest

Gamesmanship - AIDS

- Lungs Cysts = LIP (*LIP is AIDS defining in a pediatric patient*)
- Lungs Cysts + Ground Glass + Pneumothorax = PCP
- Hypervascular Nodes = Castlemans or Kaposi
- Most common airspace opacity = Strep Pneumonia
- If they show you a CT with ground glass = PCP
- “Flame Shaped” Perihilar opacity = Kaposi Sarcoma
- Persistent Opacities = Lymphoma

Infections in AIDS by CD4	
>200	Bacterial Infections, TB
<200	PCP, Atypical Mycobacterial
< 100	CMV, Disseminated Fungal, Mycobacterial

Gamesmanship: Mesothelioma

One way to show this is the “Frozen Hemithorax” - which is a lack of contralateral mediastinal shift in association with massive pleural effusion ; it’s due to encasement of the lung (and fissures) by cancer.

Gamesmanship: Collagen Vascular Tricks

RA in the shoulders on Frontal CXR = Lower Lobe UIP Pattern
 Ankylosing Spondylitis on Lateral CXR = Upper Lobe Fibrobullous Disease
 Dilated Esophagus on CT = Scleroderma with NSIP lungs

Gamesmanship: Pulmonary Edema

After the placement of a chest tube - Re-expansion Edema
 After using a bunch of crack or heroin - Drug induced Edema
 After a head injury - Neurogenic Edema
 After lung transplant - Reperfusion Edema related to ischemia/reperfusion (peak day 4)

Gamesmanship: Upper Lobes vs Lower Lobes

It's useful to have a list of what is upper lobe predominant and what is lower lobe predominant. The easiest way to ask a question would be "which of the follow is not upper lobe?" or "which of the following is upper lobe?"

Upper Lobe Predominant	Lower Lobe Predominant
Most inhaled stuff (not asbestosis). Coal Workers, and Silicosis. This includes progressive massive fibrosis.	Asbestosis
CF	Primary Ciliary Dyskinesia
RB-ILD	Most Interstitial Lung Diseases (UIP, NSIP, DIP)
Centrilobular Emphysema	Panlobular Emphysema (Alpha 1)
Ankylosing Spondylitis	Rheumatoid Lung
Sarcoid	Scleroderma (associated with NSIP)

This vs That: Pulmonary vs Mediastinal Origin

- Mediastinal Origin will make obtuse margin with lung
- Pulmonary Origin will make acute margin with lung

This vs That: Ground Glass Nodule (on PET)

- HOT GGO = Infection
- COLD GGO = Cancer (BAC)

Gamesmanship - Collapse

- * Always be on the lookout for collapse. Anytime you see anything that could be collapse at least entertain the idea.
- # Post intubation think collapse
- # Placement of central line think collapse
- * ICU patient with no other details think collapse (mucous plugging)
- Outpatient with no history think collapse (cancer).

When I Say This.....You Say That.

- When I say “obliteration of Raider’s Triangle,” you say aberrant right subclavian
- When I say “flat waist sign,” you say left lower lobe collapse
- When I say “terrorist + mediastinal widening,” you say Anthrax
- When I say “bulging fissure,” you say Klebsiella
- When I say “dental procedure gone bad, now with jaw osteo and pneumonia,” you say Actinomycosis.
- When I say “culture negative pleural effusion, 3 months later with airspace opacity,” you say TB
- When I say “hot-tub,” you say Hypersensitivity Pneumonitis
- When I say “halo sign,” you say Fungal Pneumonia - Invasive Aspergillus
- When I say “reverse halo or atoll sign,” you say COP
- When I say “finger in glove,” you say ABPA
- When I say “ABPA,” you say Asthma
- When I say “septic emboli + jugular vein thrombus,” you say Lemierre
- When I say “Lemierre,” you say Fusobacterium Necrophorum
- When I say “Paraneoplastic syndromes with SIADH,” you say Small Cell Lung CA
- When I say “Paraneoplastic syndromes with PTH,” you say Squamous Cell CA
- When I say “Small Cell Lung CA + Proximal Weakness,” you say Lambert Eaton
- When I say “Cavity fills with air, post pneumonectomy,” you say Bronchopleural Fistula
- When I say “malignant bronchial tumor,” you say carcinoid
- When I say “malignant tracheal tumor,” you say Adenoid Cystic
- When I say “AIDS patient with lung nodules, pleural effusion, and lymphadenopathy,” you say Lymphoma
- When I say “Gallium Negative,” you say Kaposi
- When I say “Thallium Negative,” you say PCP
- When I say “Macroscopic fat and popcorn calcifications,” you say Hamartoma
- When I say “Bizarre shaped cysts,” you say LCH
- When I say “Lung Cysts in a TS patient,” you say LAM
- When I say “Panlobular Emphysema - NOT Alpha 1,” you say Ritalin Lung
- When I say “Honeycombing,” you say UIP
- When I say “The histology was heterogeneous,” you say UIP
- When I say “Ground Glass with Sub pleural Sparing,” you say NSIP
- When I say “UIP Lungs + Parietal Pleural Thickening,” you say Asbestosis
- When I say “Cavitation in the setting of silicosis,” you say TB
- When I say “Air trapping seen 6 months after lung transplant,” you say Chronic Rejection / Bronchiolitis Obliterans Syndrome
- When I say “Crazy Paving,” you say PAP
- When I say “History of constipation,” you say Lipoid Pneumonia - inferring mineral oil use / aspiration.
- When I say “UIP + Air trapping,” you say Chronic Hypersensitivity Pneumonitis
- When I say “Dilated Esophagus + ILD,” = Scleroderma (with NSIP)

- When I say “Shortness of breath when sitting up,” you say Hepatopulmonary syndrome
- When I say “Episodic hypoglycemia,” you say solitary fibrous tumor of the pleura
- When I say “Pulmonary HTN with Normal Wedge Pressure,” you say Pulmonary Veno-occlusive disease.
- When I say “Yellow Nails” you say Edema and Chylous Pleural Effusions (Yellow Nail Syndrome).
- When I say “persistent fluid collection after pleural drain/tube placement,” you say Extrapleural Hematoma.
- When I say “Displaced extrapleural fat,” you say Extrapleural Hematoma.
- When I say “Massive air leak, in the setting of trauma,” you say bronchial or tracheal injury
- When I say “Hot of PET - around the periphery,” you say pulmonary infarct
- When I say “Multi-lobar collapse,” you say sarcoid
- When I say “Classic bronchial infection,” you say TB
- When I say “Panbronchiolitis,” you say tree in bud (not centrilobular or random nodules)
- When I say “Bronchorrhea,” you say Mucinous BAC

High Yield Trivia

- The tricuspid valve is the most anterior
- The pulmonic valve is the most superior
- There are 10 lung segments on the right, and 8 lung segments on the left
- If it goes above the clavicles, it's in the posterior mediastinum (cervicothoracic sign)
- Azygos Lobe has 4 layers of pleura
- Most common pulmonary vein variant is a separate vein draining the right middle lobe
- Most common cause of pneumonia in AIDS patient is Strep Pneumonia
- Most common opportunistic infection in AIDS = PCP.
- Aspergilloma is seen in a normal immune patient
- Invasive Aspergillus is seen in an immune compromised patient
- Fleischner Society Recommendations do NOT apply to patient's with known cancers
- Eccentric calcifications in a solitary pulmonary nodule pattern is considered the most suspicious.
- A part solid nodule with a ground glass component is the most suspicious morphology you can have
- Most common lung CA to present as solitary nodule
- Stage 3B lung CA is unresectable (contralateral nodal involvement; ipsilateral or contralateral scalene or supraclavicular nodal involvement, tumor in different lobes).
- The most common cause of unilateral lymphangitic carcinomatosis is bronchogenic carcinoma lung cancer invading the lymphatics

- There is a 20 year latency between initial exposure and development of lung cancer or pleural mesothelioma
- Pleural effusion is the earliest and most common finding with asbestosis exposure.
- Silicosis actually raises your risk of TB by about 3 fold.
- Nitrogen Dioxide exposure is “Silo Filler’s Disease,” gives you a pulmonary edema pattern.
- Reticular pattern in the posterior costophrenic angle is supposedly the first finding of UIP on CXR
- Sarcoidosis is the most common recurrent primary disease after lung transplant
- Pleural plaque of asbestosis typically spares the costophrenic angles.
- Pleural effusion is the most common manifestation of mets to the pleura.
- There is an association with mature teratomas and Klinefelter Syndrome.
- Injury close to the carina is going to cause a pneumomediastinum rather than a pneumothorax
- MRI is superior for assessing superior sulcus tumors because you need to look at the brachial plexus.
- Leiomyoma is the most common benign esophageal tumor (most common in the distal third).
- Esophageal Leiomyomatosis may be associated with Alport’s Syndrome
- Bronchial / Tracheal injury must be evaluated with bronchoscopy
- If you say COP also say hypersensitivity pneumonitis
- If you say BAC also say lymphoma
- Bronchial Atresia is classically in the LUL
- Pericardial cysts MUST be simple, Bronchogenic cysts don’t have to be simple
- PAP follows a rule of 1/3s post treatment; 1/3 gets better, 1/3 doesn’t, 1/3 progresses to fibrosis
- Dysphagia Lusoria presents later in life as atherosclerosis develops
- Carcinoid is COLD on PET
- Wegener’s is now called Granulomatosis with Polyangiitis - Wegener was a Nazi. Apparently he was not just a Nazi, he was a Nazi before it was “fashionable.” Plus, I heard he was a real asshole, and a bad tipper (which is unforgivable).

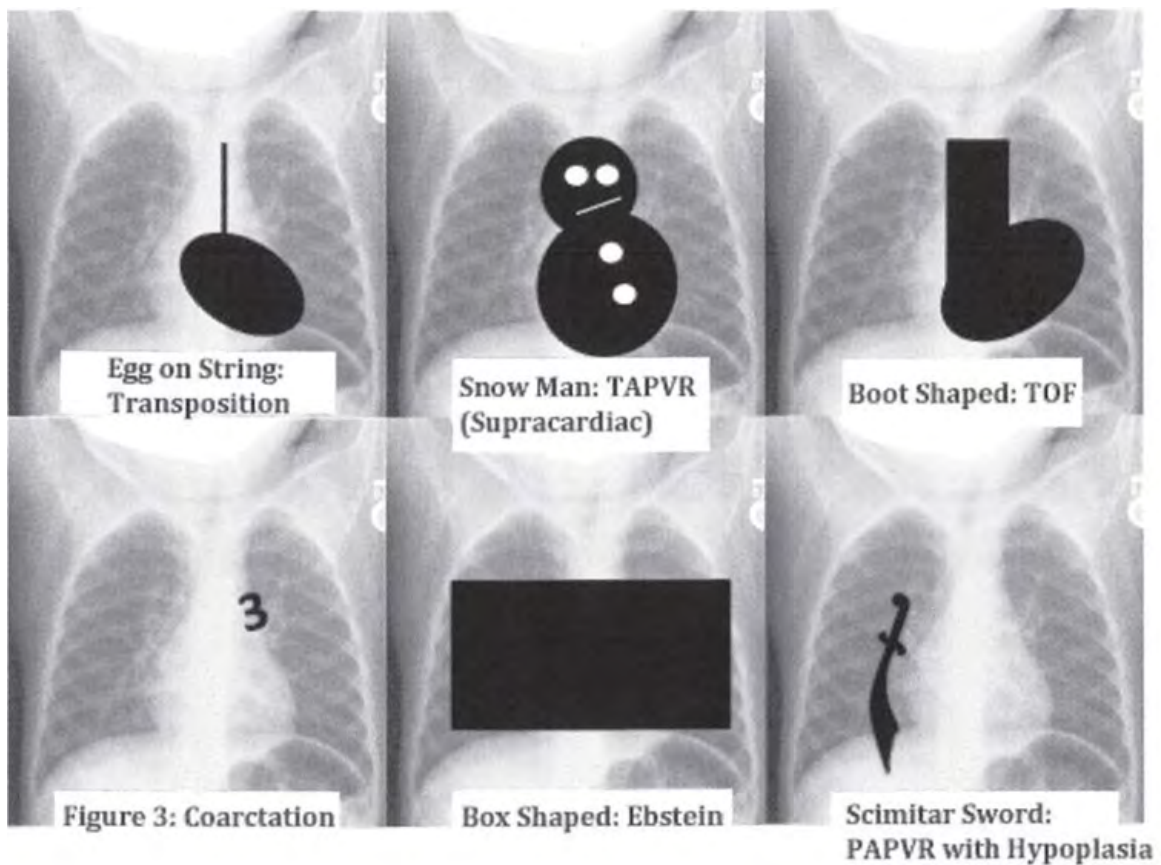
Section 6: Cardiac

Gamesmanship: Congenital Heart Disease

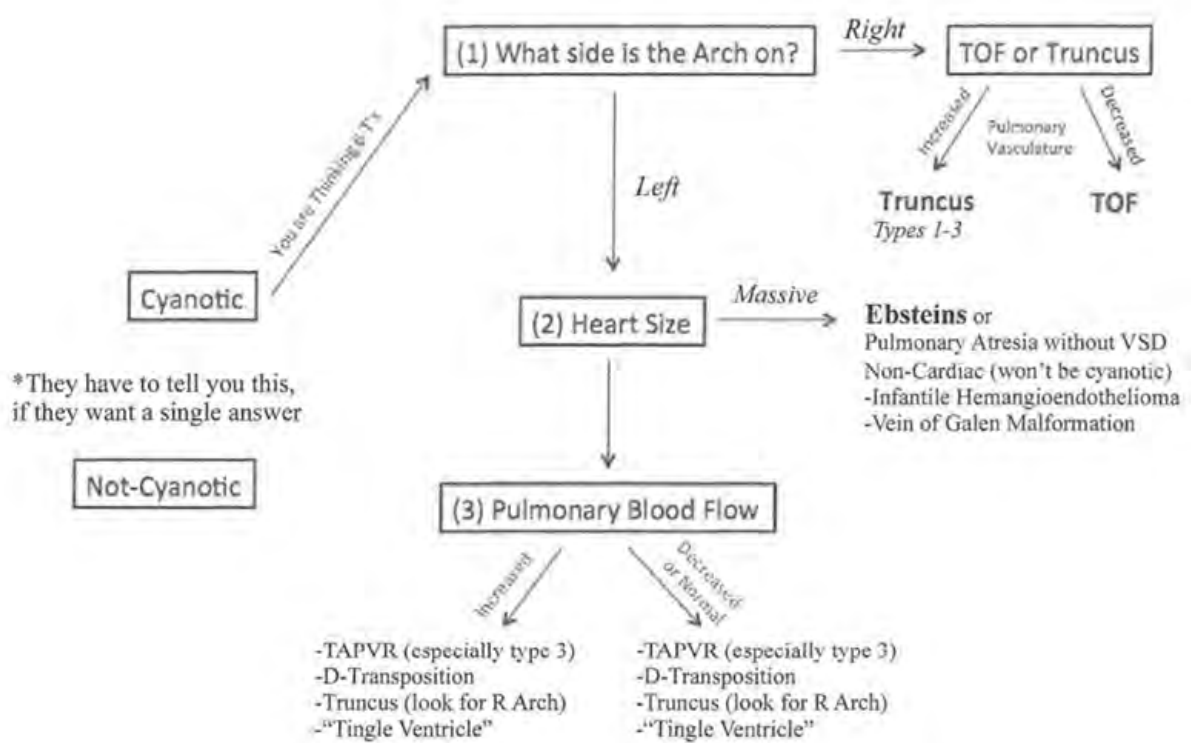
My idea is that you can only write 3 kinds of multiple choice questions regarding congenital heart disease: (1) Aunt Minnie, (2) Differential with crappy distractions, and (3) Associations/ Trivia

Situation 1: - The Aunt Minnie

There are a few congenital heart cases that are straight up Aunt Minnies. The usual characters that most 3rd year medical students memorize are fair game.



Situation 2 - Easily Solvable Cases (Bad Distractors):



Walking through this outline. First ask yourself is it cyanotic or not? They will have to tell you this in the stem. Look for this in the stem every time, then cross out answers that are not cyanotic.

Example: Patient “X” is a newborn cyanotic, what is the most likely Dx?

- A - VSD
- B-ASD
- C- Demonic Possession
- D -TOF

Cyanotic	Not Cyanotic
tof	ASD
TAPVR	VSD
transposition	PDA
Truncus	PAPVR
tricuspid Atresia	Aortic Coarctation (adult type - post ductal)

Without even looking at a picture (which they will probably show), you know the answer is D, because that is the only cyanotic one listed. If you were wondering about C - I did a google scholar search for “*Demonic Possession causing cyanosis*”, and although there were a few case reports none come down hard on cyanosis.

The next thing to ask is what side the arch is on? If you see it on the left, it's not helpful. If you see it on the right - think TOF and Truncus.

Example: Patient "X" is a newborn cyanotic, with CXR shown. What is the most likely Dx.

- A-ASD
- B-VSD
- C-Tricuspid Atresia
- D-TOF



So ASD, and VSD are out because they are not cyanotic. You notice the right arch - so you call it TOF.

Example: Patient "X" is a newborn cyanotic, with CXR shown. What is the most likely Dx.

- A- ASD
- B- VSD
- C- Truncus
- D-TOF

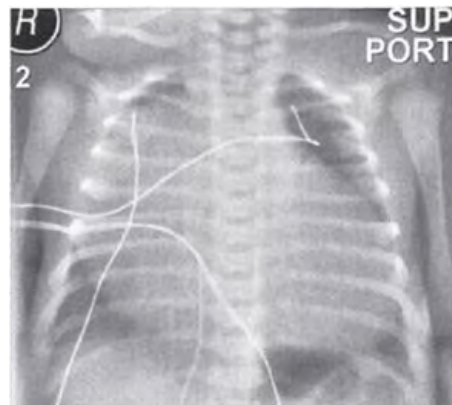
Now if they would give you the same picture but add the choice Truncus. Then you would have to look at the lungs. **Vasculature increased with Truncus, and normal / decreased with TOF.**

If they show you a normal left arch, the first thing I like to do is ask *is the heart massively enlarged?*

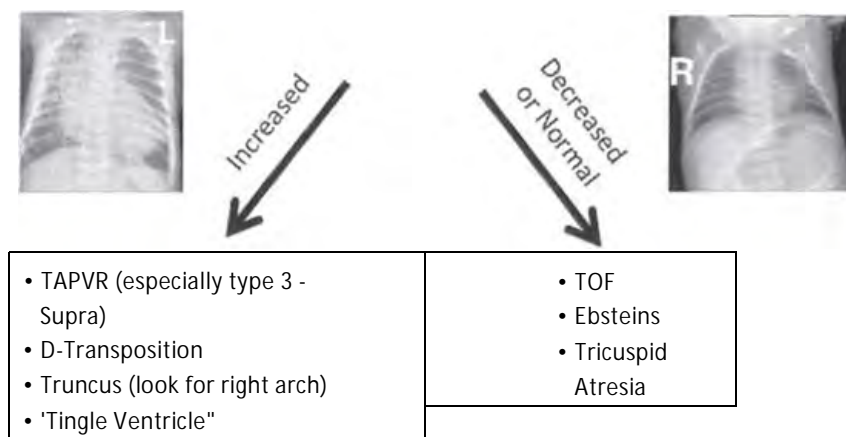
This gives you a differential of cardiac causes, and non-cardiac causes.

Cardiac: Ebstein's, or Pulmonary Atresia (without VSD).

Non- Cardiac: High Flow States like Vein of Galen Malformation and Hepatic Hemangioendothelioma. Obviously they have to show you a liver or brain next with these two.



Lastly the difference between increased and normal/decreased pulmonary vasculature can help eliminate distractors. I want to stress not trying to tell normal and decreased apart on crappy monitors. It's hard enough on a real viewing monitor. The distinction is not necessary for multiple choice.



Example: Patient with cyanotic heart disease, with CXR shown.

What is the most likely Dx?

- A - ASD
- B - VSC
- C - Tricuspid Atresia
- D - TAPVR

ASD and VSD are not cyanotic. Tricuspid Atresia has Decreased pulmonary vasculature (this CXR shows increased). That leaves TAPVR.



Gamesmanship - Signs of left atrial enlargement

- (1) Double Density - superimposed over contour of the right heart
- (2) Splaying of the carina - angle over 90 degrees
- (3) Posterior displacement of the heart - seen on the lateral CXR

Gamesmanship: Which MRI sequence is best?

Pathology	Which Sequence(s) most useful?
Cardiac Myxoma	Low T1, High T2 (high myxoid content)
Acute vs Chronic MI	Look at T2 - Bright on Acute ; Dark on Chronic (fibrous scar)
Arrhythmogenic Right Ventricular Dysplasia (ARVD)	T1 Bright
Microvascular Obstruction	First Pass Perfusion (25 seconds post Gad)
Infarct	Delayed Enhancement (10-12 mins post Gad)

This vs That: Constrictive vs Restrictive Cardiomyopathy:

- Pericardium is usually thickened in constrictive
- Diastolic septal bounce is seen in constrictive (Sigmoidization of the septum).

This vs That: True vs False Ventricular Aneurysm

- * *True:* Mouth is wider than body. Myocardium is intact. Usually anterior-lateral wall.
- * *False:* Mouth is narrow compared to body. Myocardium is NOT intact (pericardial adhesions contain rupture). Usually posterior-lateral wall. Higher risk of rupture..

This vs That: Valve Anatomy

Aortic Valve: *Right, Left, and Posterior Cusps*

Pulmonic Valve: *Right, Left, and Anterior Cusps*

This vs That: Stunned vs Hibernating Myocardium

- * *Stunned Myocardium:* After an Acute Injury (ischemia or reperfusion injury), dysfunction of myocardium persists even after restoration of blood flow (can last days to weeks). A perfusion study will be normal, but the contractility is crap.
- * *Hibernating Myocardium:* This is a more chronic process, and the result of severe CAD causing chronic hypoperfusion. You will have areas of decreased perfusion and decreased contractility even when resting. Don't get it twisted, this is not an infarct. On a FDG PET, this tissue will take up tracer more intensely than normal myocardium, and will also demonstrate redistribution of thallium. This is reversible with revascularization.
- * *Scar:* This is dead myocardium. It will not squeeze normally, so you'll have abnormal wall motion. It's not a zombie. It will not come back to life with revascularization.

Stunned	Hibernating	Infarct / Scar
Wall Motion Abnormal	Wall Motion Abnormal	Wall Motion Abnormal
Normal Perfusion (Thallium or Sestamibi)	Abnormal Fixed Perfusion	Abnormal Fixed Perfusion
	Will Redistribute with Delayed Thallium and will take up FDG	Will NOT Redistribute with Delayed Thallium, will NOT take up FDG
Associated with acute MI	Associated with chronic high grade CAD	Associated the chronic prior MI

This vs That: Left Atrial Myxoma vs Clot

- The myxoma will enhance

This vs That: Vegetations vs Fibroelastoma

You tell the difference by looking for valvular damage (seen with vegetations). Contrast enhancement is not very reliable because of how small these things are.

When I Say This.....You Say That,

- When I say “ALCAPA,” you say Steal Syndrome
- When I say “Supra-valvular Aortic Stenosis” you say Williams Syndrome
- When I say “Bicuspid Aortic Valve and Coarctation” you say Turners Syndrome
- When I say “Isolated right upper lobe edema,” you say Mitral Regurgitation
- When I say “Peripheral pulmonary stenosis,” you say Alagille Syndrome
- When I say “Box shaped heart”, you say Ebsteins
- When I say “Right Arch with Mirror Branching,” you say congenital heart.
- When I say “hand/thumb defects + ASD,” you say Holt Oram
- When I say “ostium primum ASD (or endocardial cushion defect),” you say Downs
- When I say “Right Sided PAPVR,” you say Sinus Venosus ASD
- When I say “Calcification in the left atrium wall,” you say Rheumatic Heart Disease
- When I say “difficult to suppress myocardium,” you say Amyloid
- When I say “blood pool suppression on delayed enhancement,” you say Amyloid
- When I say “septal bounce,” you say constrictive pericarditis
- When I say “ventricular interdependence,” you say constrictive pericarditis
- When I say “focal thickening of the septum - but not Hypertrophic Cardiomyopathy,” you say Sarcoid.
- When I say “ballooning of the left ventricular apex,” you say Tako-Tsubo
- When I say “fat in the wall of a dilated right ventricle,” you say Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)
- When I say “kid with dilated heart and mid wall enhancement,” you say Muscular Dystrophy
- When I say “Cardiac Rhabdomyoma,” you say Tuberous Sclerosis
- When I say “Bilateral Atrial Thrombus,” you say Eosinophilic Cardiomyopathy
- When I say “Diffuse LV Subendocardial enhancement not restricted to a vascular distribution,” you say Cardiac Amyloid.
- When I say “Glenn Procedure,” you say acquired pulmonary AVMs
- When I say “Pulmonary Vein Stenosis,” you say Ablation for A-Fib
- When I say “Multiple Cardiac Myxomas,” you say Carney’s Complex

High Yield Trivia

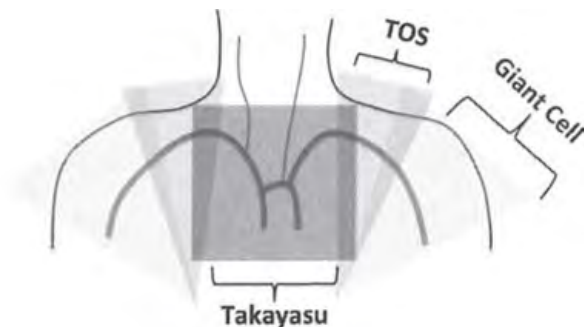
- The right atrium is defined by the I VC.
- The right ventricle is defined by the moderator band.
- The tricuspid papillary muscles insert on the septum (mitral ones do not).
- Lipomatous Hypertrophy of the Intra-Atrial Septum - can be PET Avid (it's brown fat)
- LAD gives off diagonals
- RCA gives off acute marginals
- LCX gives off obtuse marginals
- RCA perfuses SA and AV nodes (most of the time)
- Dominance is decided by which vessel lives off the posterior descending - it's the right 85%
- LCA from the Right Coronary Cusp - always gets repaired
- RCA from the Left Coronary Cusp - repaired if symptoms
- Most common location of myocardial bridging is in the mid portion of the LAD.
- Coronary Artery Aneurysm - most common cause in adult = Atherosclerosis
- Coronary Artery Aneurysm - most common cause in child = Kawasaki
- Rheumatic heart disease is the most common cause of mitral stenosis
- Pulmonary Arterial Hypertension is the most common cause of tricuspid atresia.
- Double most common vascular ring is the double aortic arch
- Most common congenital heart disease is a VSD
- Most common ASD is the Secundum
- Infracardiac TAPVR classically shown with pulmonary edema in a newborn
- "L" Transposition type is congenitally corrected (they are "L"ucky).
- "D" Transposition type is doomed.
- Truncus is associated with CATCH-22 (DiGeorge)
- Rib Notching from coarctations spares the 1st and 2nd Ribs
- Infarct with 50% involvement is unlikely to recover function
- Microvascular Obstruction is NOT seen in chronic infarct
- Amyloid is the most common cause of restricted cardiomyopathy
- Primary amyloid can be seen in multiple myeloma
- Most common neoplasm to involve the cardiac valves = Fibroelastoma
- Most commonly the congenital absence of the pericardium is partial and involves the pericardium over the left atrium and adjacent pulmonary artery (*the left atrial appendage is the most at risk to become strangulated*).
- Glenn shunt - SVC to pulmonary artery (vein to artery)
- Blalock-Taussig Shunt - Subclavian Artery to Pulmonary Artery (artery - artery)
- Ross Procedure - Replaces aortic valve with pulmonic, and pulmonic with a graft (done for kids).
- Aliasing is common with Cardiac MRI. You can fix it by: (1) opening your FOV, (2) oversampling the frequency encoding direction, or (3) switching phase and frequency encoding directions.

- * Giant Coronary Artery Aneurysms (>8mm) don't regress, and are associated with Mis.
- * Wet Beriberi (thiamine def) can cause a dilated cardiomyopathy.
- * Most common primary cardiac tumor in children = Rhabdomyoma.
- * 2nd most common primary cardiac tumor in children = Fibroma
- * Most common complication of MI is myocardial remodeling.
- * Unroofed coronary sinus is associated with Persistent left SVC.
- * Most common source of cardiac mets = Lung Cancer (lymphoma #2).
- * A-Fib is most commonly associated with left atrial enlargement
- * Most common cause of tricuspid insufficiency is RVH (usually from pulmonary FITN / cor pulmonale).

Section 7: Vascular

Gamesmanship: Thoracic Angiogram

If you see an angiogram through the great vessels and aorta think about TOS, Takayasu, and Giant Cell. The locations are classic, and helpful. Having said that remember Takayasu is going to be a young person (probably Asian female), and Giant Cell is going to be an old person - **age trumps location**. If they show you TOS, they will show the arms up and down - dead give away.



If the history is trauma, don't forget to look at the great vessels (not just the aorta).

Gamesmanship: Aortic Dissection on Angiogram

Can be shown as opacification of abdominal aortic branch vessels during aortography (catheter placed in the aortic true lumen) with the branch vessels—(celiac axis, superior mesenteric artery, and renal arteries) arising out of nowhere. They appear to be floating, with little or no antegrade opacification of the aortic true lumen. This is the so called “**floating viscera sign**.”

Gamesmanship: Collateral Filling

If you inject the SMA and the celiac branches fill - infers a tight stenosis at the celiac origin.
If you inject the celiac and the SMA branches fill - infers a tight stenosis at the SMA origin.

Gamesmanship: Hand Angiograms

Pathology: It's going to be either Buerger's or Hypothenar Hammer Syndrome(HHS).

Ask yourself is the ulnar artery involved? If yes go with the HHS. If the ulnar nerve looks ok, but the fingers are out go with Buerger's. Be careful, because the fingers can be out with HHS as well (distal emboli). Pseudo-aneurysm off the ulnar artery is a slam dunk for HHS.

Gamesmanship: Renal Artery Angiogram

Ostial Narrowing - Think Atherosclerosis - Treat with Balloon + Stent

Beading mid vessel - Think FMD - Treat with Balloon Only

Gamesmanship: Kidney Angiogram

First question should always be ? Is there an RCC or AML

Second question should be is there PAN / Speed Kidney ? A bunch of little aneurysms

Gamesmanship: Kawasaki

Two classic ways to show this: (1) CT showing a coronary artery aneurysm - the obvious one, (2) Calcified coronary artery aneurysm shown on a CXR- this was an old oral boards favorite.

This vs That: Aortic Coarctation

- # Infantile (Pre-ductal) - these guys can have pulmonary edema. More typically a long segment. Blood supply to the descending aorta is via the PDA.
- * Adult (Ductal) - Not symptomatic until later in childhood. Often presents with differential arm/leg blood pressures. More typically a short segment.

This vs That: External vs Internal Carotid

Internal Carotid	External Carotid
Low Resistance	High Resistance
Low Systolic Velocity	High Systolic Velocity
Diastolic velocity does not return to baseline	Diastolic velocity approaches zero baseline
Continuous color flow is seen throughout the cardiac cycle	Color flow is intermittent during the cardiac cycle

Also you can (1) look for branches of the external carotid, or (2) use a “temporal tap” to see ripples in the ECA spectrum.

When I Say This..... You Say That.....

- * When I say “vessel in the fissure of the ligamentum venosum,” you say replaced left hepatic artery.
- * When I say “vessel coursing of the pelvic brim,” you say Corona Mortis
- * When I say “ascending aorta calcifications,” you say Syphilis and Takayasu
- * When I say “tulip bulb aorta,” you say Marfans
- * When I say “really shitty Marfan’s variant,” you say Loeys-Dietz
- * When I say “tortuous vessels,” you say Loeys-Dietz
- * When I say “renal artery stenosis with HTN in a child,” you say NF-1
- * When I say “nasty looking saccular aneurysm, without intimal calcifications” you say Mycotic.
- * When I say “tree bark intimal calcification,” you say Syphilitic (Luetic) aneurysm
- * When I say “painful aneurysm in smoker, sparing the posterior wall,” you say Inflammatory aneurysm.
- * When I say “Turkish guy with pulmonary artery aneurysm,” you say Behcets
- * When I say “GI bleed with early opacification of a dilated draining vein,” you say Colonic Angiodysplasia
- * When I say “spider web appearance of hepatic veins on angiogram,” you say Budd Chiari
- * When I say “non-decompressible varicocele,” you say look in the belly for badness
- * When I say “right sided varicocele,” you say look in the below for badness
- * When I say “swollen left leg,” you say May Thumer
- * When I say “popliteal aneurysm,” you say look for the AAA (and the other leg)
- * When I say “most dreaded complication of popliteal aneurysm,” you say distal emboli
- * When I say “Great saphenous vein on the wrong side of the calf - lateral side,” you say Marginal Vein of Servelle - which is supposedly pathognomonic for Klippel-Trenaunay Syndrome
- * When I say “Asian,” you say Takayasu
- * When I say “Involves the aorta,” you say Takayasu
- * When I say “Kids with vertigo and aortitis,” you say Cogan Syndrome
- * When I say “Nasal perforation + Cavitory Lung Lesions,” you say Wegners
- * When I say “diffuse pulmonary hemorrhage,” you say Microscopic Polyangitis
- * When I say “Smoker + Hand Angiogram,” you say Buerger’s
- * When I say “Construction worker + Hand Angiogram,” you say Hypothenar Hammer
- * When I say “Unilateral tardus parvus in the carotid,” you say stenosis of the innominate
- * When I say “Bilateral tardus parvus in the carotids,” you say aortic stenosis
- * When I say “Bilateral reversal of flow in carotids,” you say aortic regurg
- * When I say “Lack of diastolic flow on carotid US,” you say Brain Death

High Yield Trivia

- Artery of Adamkiewicz comes off on the left side (70%) between T8-L1 (90%)
- Arch of Riolan - middle colic branch of the SMA with the left colic of the IMA.
- Most common hepatic vascular variant = right hepatic artery replaced off the SMA
- The proper right hepatic artery is anterior the right portal vein, whereas the replaced right hepatic artery is posterior to the main portal vein.
- Accessory right inferior hepatic vein - most common hepatic venous variant.
- Anterior tibialis is the first branch off the popliteal
- * Common Femoral Artery (CFA): Begins at the level of inguinal ligament
- Superficial Femoral Artery (SFA): Begins once the CFA gives off the profunda femurs
- * Popliteal Artery: Begins as the SFA exits the adductor canal
- Popliteal Artery terminates as the anterior tibial artery and the tibioperoneal trunk
- * Axillary Artery: Begins at the first rib
- * Brachial Artery: Begins as it crosses the teres major
- Brachial Artery: Bifurcates to the ulnar and radial artery
- * Intraosseous Branch: Typically arises from the ulnar
- Superficial Arch = From the Ulna, Deep Arch = From the Radius
- * The “coronary vein,” is the left gastric.
- * Enlarged splenorenal shunts are associated with hepatic encephalopathy.
- * Aortic Dissection, and intramural hematoma are caused by HTN (70%)
- * Penetrating Ulcer is from atherosclerosis.
- * Strongest predictor of progression of dissection in intramural hematoma = Maximum aortic diameter > 5cm.
- * Leriche Syndrome Triad: Claudication, Absent/ Decreased femoral pulses, Impotence.
- * Most common associated defect with aortic coarctation = bicuspid aorta (80%)
- Neurogenic compression is the most common subtype of thoracic outlet syndrome
- * Splenic artery aneurysm - More common in pregnancy, more likely to rupture in pregnancy.
- Median Arcuate Compression - worse with expiration
- # Colonic Angiodysplasia is associated with aortic stenosis
- Popliteal Aneurysm; 30-50% have AAA, 10% of patient with AAA have popliteal aneurysm, 50-70% of popliteal aneurysms are bilateral.
- Medial deviation of the popliteal artery by the medial head of the gastrocnemius = Popliteal Entrapment
- * Type 3 Takayasu is the most common (arch + abdominal aorta).
- Most common vasculitis in a kid = HSP (Henoch-Schonlein Purpura)

Section 8: IR

Gamesmanship - Self Expandable vs Balloon Expandable

Self Expandable - any where you might get external compression

Balloon Expandable - if you need more precise placement

- May Thurner Syndrome - Self Expandable
- SFA - Self Expandable
- Focal Atherosclerosis Stenosis in the Distal Aorta - Balloon Expandable
- Renal Ostium Stenosis - Balloon Expandable (needs precise placement)

This vs That -Biliary Duct Anatomy

The Right Posterior Duct drains 6&7 - runs more horizontal

The Right Anterior Duct drains 5& 8 - runs more vertical

This vs That - Dialysis

The surgically created AV Fistula has superior longevity. A fistula typically needs 3-4 months to “mature” (vein to enlarge enough for dialysis). A synthetic graft will be ready for use in 2 weeks. A synthetic graft is usually easier to declot (the clot is usually confined to the synthetic graft).

“My Leg Hurts”		
Viabile	Normal Capillary Return Normal Sensation Normal Strength	Lytic Therapy
Acute Threatened	Slow Capillary Return Minimal Sensory Loss Normal Strength	Lytic Therapy
Immediately Threatened	More Sensory Loss Mild-Moderate Weakness	Lytic Therapy ONLY if patient is a poor surgical candidate
Irreversible	Profound Sensory Loss Paralysis	Surgery

Gamesmanship - TIPS

There are several sneaky things that can be shown related to TIPS.

- CO₂ run during hepatic vein wedge- Blowing the liver dome off, because the injection was too strong. Anytime you see a CO₂ run over the liver think about this.
- TIPS placed into the hepatic artery (not portal vein). Remember to confirm that you are in the right structure - pay attention to the anatomy.
- They could tell you the portal systemic gradient was normal (3-6). Remember - that TIPS treats portal hypertension. Don't do a TIPS on someone who does NOT have portal HTN.

When I Say This.....You Say That.....

- When I say "Hairpin turn - during bronchial angiography," you say anterior medullary (spinal cord) artery
- When I say "Fever, WBC, Nausea, and Vomiting after Uterine Artery Embolization," you say Post Embolization Syndrome (obviously could also be infection)
- When I say "Most medial vessel in the leg," you say posterior tibial
- When I say "the source of 85% of upper GI bleeds," you say left gastric
- When I say "the source of bleeding from a duodenal ulcer," you say GDA
- When I say "Pulmonary AVM," you say HHT
- When I say "most feared complication of bronchial artery embolization," you say spinal cord infarct
- When I say "high risk of bleeding for liver transplant," you say transjugular approach
- When I say "most feared complication of brachial arterial access," you say compartment syndrome
- When I say "cold painful fingers during dialysis," you say "Steal syndrome"
- When I say "ulcer on medial ankle," you say venous stasis
- When I say "ulcer on dorsum of foot," you say ischemia or infected ulcer
- When I say "ulcer on plantar surface of foot," you say neutropenic ulcer

High Yield Trivia

- “Significant lesion” = A systolic pressure gradient > 10 mm Hg at rest
- Things to NOT stick a drain in: Tumors, Acute Hematoma, and those associated with acute bowel rupture and peritonitis
- Renal Artery Stenting for renal failure - tends to not work if the Cr is > 3 .
- Persistent sciatic artery is prone to aneurysm
- Even if the cholecystostomy tube instantly resolves all symptoms, you need to leave the tube in for 2-6 weeks (until the tract matures), otherwise you are going to get a bile leak.
- MELD scores greater than 24 are at risk of early death with TIPS
- The target gradient post tips (for esophageal bleeding) is between 9 and 11.
- Absolute contraindication for TIPS - Heart Failure, Severe Hepatic Failure
- Most common side effect of BRTO is gross hematuria
- Sensitivity = GI Bleed Scan = 0.1 mL/min , Angiography = 1 .OmL/min
- For GI Bleed - after performing an embolization of the GDA (for duodenal ulcer), you need to do a run of the SMA to look at the inferior pancreaticoduodenal
- Most common cause of lower GI bleed is diverticulosis
- TACE will prolong survival better than systemic chemo
- TACE: Portal Vein Thrombosis is considered a contraindication (sometimes) because of the risk of infracting the liver.
- Go above the rib for Thora
- Left Bundle Branch Block needs a pacer before a Thoracic Angiogram
- Never inject contrast through a swan ganz catheter for a thoracic angiogram
- You treat pulmonary AVMs at 3mm
- Hemoptysis - Active extravasation is NOT typically seen with the active bleed.
- UAE - Gonadotropin-releasing medications (often prescribed for fibroids) should be stopped for 3 months prior to the case
- The general rule for transluteal is to avoid the sciatic nerves and gluteal arteries by access through the sacrospinous ligament medially (close to the sacrum, inferior to the piriformis).
- When to pull an abscess catheter; As a general rule - when the patient is better (no fever, WBC normal), and output is < 20 cc over 24 hours.
- If the thyroid biopsy is non-diagnostic, you have to wait 3 months before you re-biopsy.
- Posterior lateral approach is the move for percutaneous nephrostomy
- You can typically pull a sheath with an ACT < 150 -180
- Notice that 0.039, 0.035, 0.018 wires are in INCHES
- 3 French = 1 mm
- French size is the OUTSIDE of a catheter and the INSIDE of a sheath
- Artery calcifications (common in diabetics) make compression difficult, and can lead to a false elevation of the ABI.

- Type 2 endoleaks are the most common
- Circumaortic left renal vein: the anterior one is superior, the posterior one is inferior, and the filter should be below the lowest one.
- Risk of DVT is increased with IVC filters
- Acute Budd Chiari with fulminant liver failure = Needs a TIPS
- Pseudoaneurysm of the pancreaticoduodenal artery = “Sandwich technique” - distal and proximal segments of the artery feeding off the artery must be embolized
- Median Arcuate Ligament Syndrome - First line is surgical release of the ligament
- Massive Hemoptysis = Bronchial artery - Particles bigger than 325 micrometers
- Acalculous Cholecystitis = Percutaneous Cholecystostomy
- Hepatic encephalopathy after TIPS = You can either (1) place a new covered stent constricted in the middle by a loop of suture - deployed in the pre-existing TIPS, (2) place two new stents - parallel to each other (one covered self expandable, one uncovered balloon expandable).
- Recurrent variceal bleeding after placement of a constricted stent - balloon dilation of the constricted stent
- Appendiceal Abscess - Drain placement * just remember that a drain should be used for a mature (walled off) abscess and no frank peritoneal symptoms
- Inadvertent catheterization of the colon (after trying to place a drain in an abscess) - wait 4 weeks for the tract to mature - verify by over the wire tractogram, and then remove tube.
- DVT with severe symptoms and no response to systemic anticoagulation = Catheter Directed Thrombolysis

Section 9: Nukes

Tracer	Analog	Energy	Physical Half Life
Tc - 99m		"Low" - 140	6 hours
Iodine -123	Iodine	"Low" - 159	13 hours
Xenon - 133		"Low"-81	125 hours (<i>biologic tl/2 30 seconds</i>)
Thallium - 201	Potassium	"Low"-135 (2%), 167 (8%), <i>use 71 ²⁰¹ Hg daughter x-rays</i>	73 hours
Indium - 111		"Medium"- 173 (89%), 247 (94%)	67 hours
Gallium - 67	Iron	Multiple; 93 (40%), 184 (20%), 300 (20%), 393 (5%)	78 hours
Iodine -131	Iodine	"High" - 365	8 days
Fluorine -18	Sugar	"High"-511	110 mins

Treatment Radionuclides Half Life	
Strontium 89	50.5 DAYS (14 days in bone)
Samarium 153	46 Hours
Yttrium 90	64 Hours

Gamesmanship: - Tc-99 DTPA vs Xe-133

Distinguishing these two: The DTPA can be done in multiple projections. The DTPA tends to clump in the central airways.

This vs That - Tc WBC vs In WBC

Tc WBC	In WBC
Renal	NO Renal
GI	NO GI

This vs That - Tc WBC at 4 hours, Tc WBC at 24 hours

4 hours - lung uptake

24 hours - lung uptake has cleared, start to get bowel uptake

This vs That - Tc MDP vs F-18 Bone Scan - organ with higher dose

Tc MDP - Bone

F-18 - Bladder

This vs That - Bone Met Therapy

Sr ⁸⁹	Sm ¹⁵³	Ra ²²³
Pure Beta Emitter	Beta Emitter, with imagable some gamma	Alpha Emitter
Most Bone Marrow Toxicity (longest recovery).	Less Bone Marrow Toxicity	Least Bone Marrow Toxicity
Renal excretion	Renal excretion	GI excretion
		Improves Survival (prostate mets)

This vs That - Renal Tracer Mechanisms

Tc DTPA	Tc MAG 3	Tc GH
Filtered (GFR)	Secreted (ERPF)	Filtered
Good For Native Kidneys with Normal Renal Function	Concentrated better by kidneys with poor renal function	Good for dynamic and cortical imaging.
Critical Organ Bladder	Critical Organ Bladder	Critical Organ Bladder

This vs That - ATN vs Rejection vs Drug Tox

ATN	Immediate Post OP (3-4 days post op)	Perfusion Normal	Excretion Delayed
Cyclosporin Toxicity	Long Standing	Perfusion Normal	Excretion Delayed
Acute Rejection	Immediate Post OP	Poor Perfusion	Excretion Delayed

This vs That - Cancer vs Maybe Not Cancer (clinical correlation)

Tumors that are PET COLD	Not Cancer but PET HOT
BAC (Adeno In Situ) - Lung Cancer	Infection
Carcinoid	Inflammation
RCC	Ovaries in Follicular Phase
Peritoneal Bowel/Liver Implants	Muscles
Anything Mucinous	Brown Fat
Prostate	Thymus

This vs That - Why is Granny is confused?

FDG PET - Brain		
Alzheimer	Low posterior temporoparietal cortical activity	Identical to Parkinson Dementia
Multi Infarct	Scattered areas of decreased activity	
Dementia with Lewy Bodies	Low in lateral occipital cortex	Preservation of the mid posterior cingulate gyrus (Cingulate Island Sign)
Picks / Frontotemporal	Low frontal lobe	
Huntingtons	Low activity in caudate nucleus and putamen	

This vs That - Graves vs Toxic Multi-Nodular Goiter

Graves	Toxic Multi-Nodular Goiter
Uptake High :70s	Uptake Medium High: 40s
Homogenous	Heterogeneous

When I Say This.....You Say That.....

- When I say “hot clumps of signal in the lungs on Liver Spleen sulfur colloid,” you say too much Al in the Tc.
- When I say “HOT spleen,” you say WBC scan or Octreotide (sulfur colloid will be like warm spleen).
- When I say “Bone Scan with Hot Skull Sutures,” you say renal osteodystrophy
- When I say “Bone Scan with Focal Breast Uptake,” you say breast CA
- When I say “Bone Scan with Renal Cortex Activity,” you say hemochromatosis
- When I say “Bone Scan with Liver Activity,” you say either too much Al, Amyloid, Hepatoma, or Liver Necrosis
- When I say “Bone Scan with Sternal Lesion,” you say breast CA.
- When I say “Bone Scan with Diffusely Decreased Bone Uptake,” you say (1) Free Tc, or (2) Bisphosphonate Therapy.
- When I say “Tramline along periosteum of long bones,” you say lung CA
- When I say “Super Hot Mandible in Adult,” you say Fibrous Dysplasia
- When I say “Super Hot Mandible in Child,” you say Caffey's
- When I say “Periarticular uptake of delayed scan,” you say RSD
- When I say “Focal uptake along the lesser trochanter,” you say Prosthesis loosening
- When I say “Tracer in the brain on a VQ study,” you say Shunt
- When I say “Tracer over the liver on Ventilation with Xenon,” you say Fatty Liver
- When I say “Gallium Negative, Thallium Positive,” you say Kaposi
- When I say “High T3, High T4, low TSH, - low thyroid uptake,” you say Quervain's (Granulomatous thyroiditis).
- When I say “persistent tracer in the lateral ventricles > 24 hours,” you say NPH
- When I say “Renal uptake on sulfur colloid,” you say CHF
- When I say “Renal transplant uptake on sulfur colloid”, you say Rejection
- When I say “Filtered Renal Agent,” you say DTPA (or GH)
- When I say “Secreted Renal Agent,” you say MAG-3
- When I say “PET with increased muscle uptake,” you say insulin
- When I say “Diffuse FDG uptake in the thyroid on PET,” you say Hashimoto
- When I say “I see the skeleton on MIBG,” you say diffuse neuroblastoma bone mets
- When I say “Cardiac tissue taking up FDG more intense than normal myocardium,” you say hibernating myocardium
- I say “made with a generator” , you say Tc99 and Rubidium

High Yield Trivia

- Geiger Mueller - maximum dose it can handle is about 1 OOmR/h
- Activity level greater than 100 mCi of Tc-99m is considered a major spill.
- Activity level greater than 100 mCi of Tl-201 is considered a major spill.
- Activity level greater than 10 mCi of In-111, is considered to represent a major spill.
- Activity level greater than 10 mCi of Ga-67, is considered to represent a major spill.
- An activity level greater than 1 mCi of I-131 is considered to constitute a major spill.
- Annual Dose limit of 100 mrem to the public
- Not greater than 2 mrem per hour - in an “unrestricted area”
- Total Body Dose per Year = 5 rem
- Dose to the Ocular Lens per year = 15 rem
- Total equivalent organ dose (skin is also an organ) per year = 50 rem
- Total equivalent extremity dose per year = 50 rem (500mSv)
- Total Dose to Embryo/fetus over entire 9 months - 0.5rem
- NRC allows no more than 0.15 micro Ci of Mo per 1 mili Ci of Tc, at the time of administration.
- Chemical purity (A1 in Tc) is done with pH paper
- The allowable amount of A1 is < 10 micrograms
- Radiochemical purity (looking for Free Tc) is done with thin layer chromatography
- Free Tc occurs from - lack of stannous ions or accidental air injection (which oxidizes)
- Prostate Cancer bone mets are uncommon with a PSA less than 10 mg/ml
- Flair Phenomenon occurs 2 weeks - 3 months after therapy
- Skeletal Survey is superior (more sensitive) for lytic mets
- AVN - Early and Late is COLD, Middle (repairing) is Hot.
- Particle size for VQ scan is 10-100 micrometers
- Xenon is done first during the VQ scan
- Amiodarone - classic thyroid uptake blocker
- Hashimotos increases risk for lymphoma
- Hot nodule on Tc, shouldn't be considered benign until you show that it's also hot on I¹²³. This is the concept of the discordant nodule.
- History of methimazole treatment (even years prior) makes I-131 treatment more difficult
- Methimazole side effect is neutropenia
- In pregnancy PTU is the blocker of choice
- Sestamibi in the parathyroid depends on blood flow and mitochondria
- You want to image with PET - following therapy at interval of 2-3 weeks for chemotherapy, and 8-12 weeks for radiation is the way to go. This avoids “stunning” - false negatives, and inflammatory induced false positive.

- ^{111}In Pentetreotide is the most commonly used agent for somatostatin receptor imaging. The classic use is for carcinoid tumors
- Meningiomas take up octreotide
- Prior to MIBG you should block the thyroid with Lugol's Iodine or Perchlorate
- Left bundle branch block can cause a false positive defect in the ventricular septum (spares the apex)
- Pulmonary uptake of Thallium is an indication of LV dysfunction
- MIBG mechanism is that of an Analog of Norepinephrine - actively transported and stored in the neurosecretory granules
- MDP mechanism is that of a Phosphate analog - which works via Chemisorption
- Sulfur Colloid mechanism = Particles are Phagocytized by RES

SECTION 10: NEURO

Foramen	Contents
Foramen Ovale	CN V3, and Accessory Meningeal Artery
Foramen Rotundum	CN V2 ("R2V2"),
Superior Orbital Fissure	CN 3, CN 4, CN VI, CN6
Inferior Orbital Fissure	CN V2
Foramen Spinosum	Middle Meningeal Artery
Jugular Foramen	Jugular Vein, CN 9, CN 10, CN 11
Hypoglossal Canal	CN12
Optic Canal	CN 2, and Ophthalmic Artery

This vs That: *HIV Encephalitis vs PML*

- HIV Encephalitis is symmetric (T2 bright, T1 normal)
- PML is asymmetric (T2 bright, T1 dark)

This vs That: AIDS Infections

AIDS Encephalitis	PML	CMV	Toxo	Cryptococcus
Symmetric T2 Bright	Asymmetric T2 Bright	Periventricular T2 Bright	Ring Enhancement	Dilated Perivascular Spaces
	T1 dark	Ependymal Enhancement	Thallium Cold	Basilar Meningitis

This vs That: *Toxo vs Lymphoma*

Toxo	Lymphoma
Ring Enhancing	Ring Enhancing
Hemorrhage more common after treatment	Hemorrhage less common after treatment
Thallium Cold	Thallium HOT
PET Cold	Pet Hot
MR Perfusion: Decreased CBV	MR Perfusion: Increased (or Decreased) CBV

This vs That: *LeForts Unique Components*

- LeFort 1: Lateral Nasal Aperture
- LeFort 2: Inferior Orbital Rim, and Orbital Floor
- LeFort 3: Zygomatic Arch, and Lateral Orbital Rim/Wall

This vs That: Temporal Bone Fractures:

Longitudinal	Transverse
Long Axis of T-Bone	Short Axis of T-Bone
More Common	Less Common
More Ossicular Dislocation	More Vascular Injury (Carotid / Jugular)
Less Facial Nerve Damage (around 20%)	More Facial Nerve Damage (>30%)
More Conductive Hearing Loss	More Sensorineural Hearing Loss

This vs That: *Porencephalic Cyst vs Open Lip Schizencephaly*

- Open Lip Schizencephaly - cleft lined by gray matter (malformation)
- Porencephalic Cyst - hole from prior ischemia

This vs That: Vocal Cord Paralysis vs Cancer

- Affected side is dilated with vocal cord paralysis
- Opposite side is dilated with cancer

This vs That: Syndromes with Tumors

NF-1	Optic Nerve Gliomas
NT-2	MSME; Multiple Schwannomas, Meningiomas, Ependymomas
VHL	Hemangioblastoma (brain and retina)
TS	Subependymal Giant Cell Astrocytoma, Cortical Tubers
Nevoid Basal Cell Syndrome (Gorlin)	Medulloblastoma
Turcot	GBM, Medulloblastoma
Cowdens	Lhermitte-Duclos (Dysplastic cerebellar gangliocytoma)

This vs That: Meningioma vs Schwannoma

Meningioma	Schwannoma
Enhance Homogeneously	Enhance Less Homogeneously
Don't Usually Invade IAC	Invade IAC
Calcify more often	IAC can have "trumpeted" appearance

This vs That: Where'd all that blood come from?

Maximum Bleeding-Aneurysm Location

ACOM	Interhemispheric Fissure
PCOM	Ipsilateral Basal Cistern
MCA Trifurcation	Sylvian Fissure
Basilar Tip	Interpeduncular Cistern, or Intraventricular
PICA	Posterior Fossa or Intraventricular

When I Say This.....You Say That

- When I say “cervical kyphosis” , you say NF-1
- When I say “lateral thoracic meningocele,” you say NF-1
- When I say “bilateral optic nerve gliomas,” you say NF-1
- When I say “bilateral vestibular schwannoma,” you say NF-2
- When I say “retinal hamartoma,” you say TS
- When I say “retinal angioma,” you say VHL
- When I say “brain tumor with restricted diffusion,” you say lymphoma
- When I say “brain tumor crossing the midline,” you say GBM (or lymphoma)
- When I say “Cyst and Nodule in Child,” you say Pilocystic Astrocytoma
- When I say “Cyst and Nodule in Adult,” you say Hemangioblastoma
- When I say “multiple hemangioblastoma,” you say Von Hippel Lindau
- When I say “Swiss cheese tumor in ventricle,” you say central neurocytoma
- When I say “CN3 Palsy,” you say posterior communicating artery aneurysm
- When I say “CN6 Palsy,” you say increased ICP
- When I say “Ventricles out of size to atrophy,” you say NPH
- When I say “Hemorrhagic putamen,” you say Methanol
- When I say “Decreased FDG uptake in the lateral occipital cortex,” you say Lewy Body Dementia
- When I say “TORCH with Periventricular Calcification,” you say CMV
- When I say “TORCH with hydrocephalus,” you say Toxoplasmosis
- When I say “TORCH with hemorrhagic infarction,” you say HSV
- When I say “Neonatal infection with frontal lobe atrophy,” you say HIV
- When I say “Rapidly progressing dementia + Rapidly progressing atrophy,” you say CJD
- When I say “Expanding the cortex,” Oligodendroglioma
- When I say “Tumor acquired after trauma (LP),” you say Epidermoid
- When I say “The Palate Separated from the Maxilla / Floating Palate,” you say LeFort 1
- When I say “The Maxilla Separated from the Face” or “Pyramidal” you say LeFort 2
- When I say “The Face Separated from the Cranium,” you say LeFort 3
- When I say “Airless expanded sinus,” you say mucocoele
- When I say “DVA,” you say cavernous malformation nearby
- When I say “Single vascular lesion in the pons,” you say Capillary Telangiectasia
- When I say “Elevated NAA peak,” you say Canvans
- When I say “Tigroid appearance,” you say Metachromatic Leukodystrophy
- When I say “Endolymphatic Sac Tumor,” you say VHL
- When I say “T1 Bright in the petrous apex,” you say Cholesterol Granuloma
- When I say “Restricted diffusion in the petrous apex,” you say Cholesteatoma
- When I say “Lateral rectus palsy + otomastoiditis,” you say Grandenigo Syndrome
- When I say “Cochlea and semicircular canal enhancement,” you say Labrinthitis
- When I say “Conductive hearing loss in an adult,” you say Otosclerosis
- When I say “Noise induced vertigo,” you say Superior Semicircular Canal dehiscence

- When I say “Widening of the maxillary ostium,” you say Antrochonal Polyp
- When I say “Inverting papilloma,” you say squamous cell CA (10%)
- When I say “Adenoid cystic,” you say perineural spread
- When I say “Left sided vocal cord paralysis,” you say look in the AP window
- When I say “Bilateral coloboma,” you say CHARGE syndrome
- When I say “Retinal Detachment + Small Eye” you say PHPV
- When I say “Bilateral Small Eye,” you say Retinopathy of Prematurity
- When I say “Calcification in the globe of a child,” you say Retinoblastoma
- When I say “Fluid-Fluid levels in the orbit,” you say Lymphangioma
- When I say “Orbital lesion, worse with Valsalva,” you say Varix
- When I say “Pulsatile Exophthalmos,” you say NF-1 and CC Fistula
- When I say “Sphenoid wing dysplasia,” you say NF-1
- When I say “Simitar Sacrum,” you say Currarino Triad
- When I say “bilateral symmetrically increases T2 signal in the dorsal columns,” you sat B12 (or HIV)
- When I say “Owl eye appearance of spinal cord,” you say spinal cord infarct
- When I say “Enhancement of the nerves root of the cauda equina,” you say Guillain Barre
- When I say “Subligamentous spread of infection,” you say TB

High Yield Trivia

- The order of tumor prevalence in NF2 is the same as the mnemonic MSME (schwannoma > meningioma > ependymoma).
- Maldeveloped draining veins is the etiology of Sturge Weber
- All phakomatosis (NF 1, NF -2, TS, and VHL) EXCEPT Sturge Weber are autosomal dominant - family screening is a good idea.
- Most Common Primary Brain Tumor in Adult = Astrocytoma
- "Calcifies 90% of the time" = Oligodendroglioma
- Restricted Diffusion in Ventricle = Watch out for Choroid Plexus Xanthogranuloma (not a brain tumor, a benign normal variant)
- Pituitary - T1 Bright = Pituitary Apoplexy
- Pituitary - T2 Bright = Rathke Cleft Cyst
- Pituitary - Calcified = Craniopharyngioma
- CP Angle - Invades Internal Auditory Canal = Schwannoma
- CP Angle - Invades Both Internal Auditory Canals = Schwannoma with NF2
- CP Angle - Restricts on Diffusion = Epidermoid
- Peds - Arising from Vermis = Medulloblastoma
- Peds- 4th ventricle "tooth paste" out of 4th ventricle = Ependymoma
- Adult myelination pattern: T1 at 1 year, T2 at 2 years
- Brainstem and posterior limb of the internal capsule are myelinated at birth.
- CN2 and CNV3 are not in the cavernous sinus
- Persistent trigeminal artery (vertebral to carotid) increases the risk of aneurysm
- Subfalcine herniation can lead to ACA infarct
- ADEM lesions will NOT involve the calloso-septal interface.
- Marchiafava-Bignami progresses from body -> genu -> splenium
- Post Radiation changes don't start for 2 months (there is a latent period).
- Hippocampal atrophy is first with Alzheimer Dementia
- Most common TORCH for CMV
- Toxo abscess does NOT restrict diffusion
- Small cortical tumors can be occult without IV contrast
- JPA and Ganglioglioma can enhance and are low grade
- Nasal Bone is the most common fracture
- Zygomaticomaxillary Complex Fracture (Tripod) is the most common fracture pattern and involves the zygoma, inferior orbit, and lateral orbit.
- Supplemental oxygen can mimic SAH on FLAIR
- Putamen is the most common location for hypertensive hemorrhage
- Restricted diffusion without bright signal on FLAIR should make you think hyperacute (< 6 hours) stroke.
- Enhancement of a stroke; Rule of 3s - starts at day 3, peaks at 3 weeks, gone at 3 months
- PAN is the Most Common systemic vasculitis to involve the CNS
- Scaphocephaly is the most common type of cranosynostosis

- Piriform aperture stenosis is associated with hypothalamic pituitary adrenal axis issues.
- Cholesterol Granuloma is the most common primary petrous apex lesion
- Large vestibular aqueduct syndrome has absence of the bony modiolus in 90% of cases
- Octreotide scan will be positive for esthesioneuroblastoma
- The main vascular supply to the posterior nose is the sphenopalatine artery (terminal internal maxillary artery).
- Warthins tumors take up pertechnetate
- Sjogrens gets salivary gland lymphoma
- Most common intra-ocular lesion in an adult = Melanoma
- Enhancement of nerve roots for 6 weeks after spine surgery is normal. After that it's arachnoiditis
- Hemorrhage in the cord is the most important factor for outcome in a traumatic cord injury.
- Currarino Triad: Anterior Sacral Meningocele, Anorectal malformation, Sacrococcygeal osseous defect
- Type 1 Spinal AVF (dural AVF) is by far the more common.
- Herpes spares the basal ganglia (MCA infarcts do not)

Section 11: MSK

This vs That: Forearm Fractures:

Essex-Lopresti	Galeazzi Fracture (MUGR)	Monteggia Fracture (MUGR)
Fracture of the radial head + Anterior dislocation of the distal radial ulnar joint	Radial Shaft fracture, with anterior dislocation of the ulna at the DRUJ.	Fracture of the proximal ulna, with anterior dislocation of the radial head.

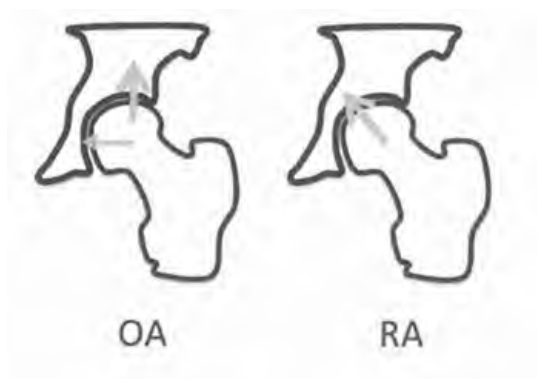
This vs That: Femoral Neck Stress Fractures

- Medial Side - Stress Fracture, Compressive Side, Dose Well
- Lateral Side - Risperphosphate, Tensile Side, Dose Terrible

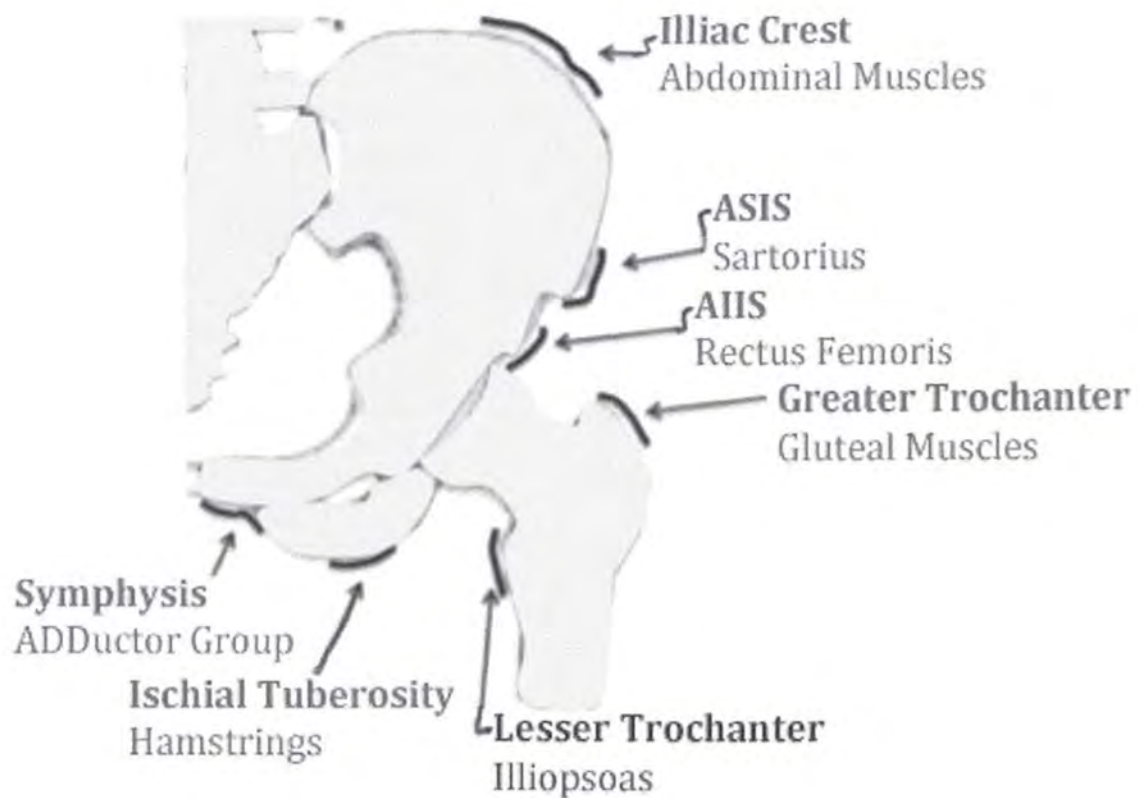
This vs That: ACL Repair Complication

- Femoral Tunnel maintains tension on the graft (Isometry)
- Tibial Tunnel prevents root impingement

This vs That: RA vs OA in the Hip



This vs That: Avulsion from where?



This vs That: Impingement Types

Pincer Impingement	Cam Impingement
Middle Aged Women	Young Man
Over Coverage of the femoral head by the acetabulum	Bony protrusion on the antero-superior femoral head-neck junction
“Cross Over Sign ”	“Pistol Grip Deformity” Describes the appearance of the femur

This vs That: Osteochondroses

Kohlers	Tarsal Navicular	Male 4-6. Treatment is not surgical.
Freiberg Infraction	Second Metatarsal Head	Adolescent Girls - can lead to secondary OA
Severs	Calcaneal Apophysis	Some say this is a normal “growing pain”
Panners	Capitellum	Kid 5-10 “Thrower” ; does not have loose bodies.
Perthes	Femoral Head	White kid; 4-8.
Kienbock	Carpal Lunate	Associated with negative ulnar variance. Seen in person 20-40.

Gamesmanship - Wrist Compartments

Gamesmanship Wrist Compartments	
Isolated 1st	De Quervains
1 st + 2nd	Intersecting
Isolated 6th	Early RA
Multiple Flexors	RA

This vs That - Finger Tumors

Finger Tip Tumors / Masses		
Glomus	T1 Dark, T2 Bright, Enhances avidly.	T2 Bright, Enhance Avidly.
Giant Cell Tumor Tendon	T1 Dark, T2 Dark, Variable Enhancement, Bloom on Gradient	Bloom on Gradient
Fibroma	T1 Dark, T2 Dark. No Blooming	Does NOT Bloom on Gradient.

When I Say This.....You Say That.....

- When I say, “Posterior elbow dislocation,” you say Capitellum fracture
- When I say “Chondroblastoma in an adult”, you say “Clear Cell Chondrosarcoma”
- When I say “Malignant epiphyseal lesion”, you say “Clear Cell Chondrosarcoma”
- When I say “Permeative lesion in the diaphysis of a child” , you say “Ewings”
- When I say “T2 bright lesion in the sacrum” , you say “Chordoma”
- When I say “Lytic T2 DARK lesion” , you say “Fibrosarcoma”
- When I say “Sarcomatous transformation of an infarct”, you say “MFH”
- When I say, “Epiphyseal Lesion that is NOT T2 Bright” , You say Chondroblastoma
- When I say, “short 4th metacarpal,” You say pseudopseudohypoparathyroidism and Turner Syndrome
- When I say, “band like acro-osteolysis,” You say Hajdu-Cheney
- When I say “fat containing tumor in the retroperitoneum,” you say liposarcoma
- When I say “sarcoma in the foot” you say synovial sarcoma.
- When I say “avulsion of the lesser trochanter,” you say pathologic fracture
- When I say “cross over sign,” you say pincher type Femoroacetabular Impingement
- When I say “Segond Fracture,” you say ACL tear
- When I say “Reverse Segond Fracture,” you say PCL
- When I say “Arcuate Sign,” you say fibular head avulsion or PCL tear
- When I say “Deep Intercondylar Notch,” you say ACL tear
- When I say “Bilateral Patellar Tendon Ruptures,” you say chronic steroids
- When I say “Wide ankle mortise,” you say show me the proximal fibula (Maisonneuve).
- When I say “Bilateral calcaneus fractures,” you say show me the spinal compression fracture (“lover’s leap”)
- When I say “Dancer with lateral foot pain,” you say avulsion of 5th MT
- When I say “Old lady with sudden knee pain with standing,” you say SONK
- When I say “Looser’s Zones,” you say osteomalacia or rickets (vitamin D)
- When I say “Unilateral RA with preserved joint spaces,” you say RSD
- When I say “T2 bright tumor in finger,” you say Glomus
- When I say “Blooming in tumor in finger,” you say Giant Cell Tumor of Tendon Sheath (PVNS)
- When I say “Atrophy of teres minor,” you say Quadrilateral Space syndrome
- When I say “Subluxation of the Biceps Tendon,” you say Subscapularis tear
- When I say “Too many bow ties,” you say Discoid Meniscus
- When I say “Celery Stalk ACL - T2” you say Muroid Degeneration
- When I say “Drumstick ALC - T1” you say Muroid Degeneration
- When I say “Acute Flat foot,” you say Posterior Tibial Tendon Tear
- When I say “Boomerang shaped peroneus brevis,” you say tear - or split tear

- When I say “Meniscoid mass in the lateral gutter of the ankle,” you say Anteriolateral Impingement Syndrome
- When I say “Scar between 3rd and 4th metatarsals,” you say Morton’s neuroma
- When I say “Osteomyelitis in the spine,” you say IV drug user
- When I say “Osteomyelitis in the spine with Kyphosis,” you say TB (Gibbus Deformity)
- When I say “Unilateral SI joint lysis,” you say IV Drug User
- When I say “Psoas muscle abscess,” you say TB
- When I say “Rice bodies in joint,” you say TB - sloughed synovium
- When I say “Calcification along the periphery,” you say myositis ossificans
- When I say “Calcifications more dense in the center,” you say Osteosarcoma - reverse zoning
- When I say “Permeative lesion in the diaphysis of a child,” you say Ewings
- When I say “Long lesion in a long bone,” you say Fibrous Dysplasia
- When I say “Large amount of edema for the size of the lesion,” you say Osteoid Osteoma
- When I say “Cystic bone lesion, that is NOT T2 bright,” you say Chondroblastoma
- When I say “Lesion in the finger of a kid,” you say Periosteal chondroma
- When I say “looks like NOF in the anterior tibia with anterior bowing,” you say Osteofibrous Dysplasia.
- When I say “ RA + Pneumoconiosis,” you say Caplan Syndrome
- When I say “ RA + Big Spleen + Neutropenia,” you say Felty Syndrome
- When I say “Reducible deformity of joints - in hand,” you say Lupus.
- When I say “destructive mass in a bone of a leukemia patient,” you say Chloroma

High Yield Trivia

- Arthritis at the radioscaphoid compartment is the first sign of a SNAC or SLAC wrist
- SLAC wrist has a DISI deformity
- The pull of the Abductor pollicis longus tendon is what causes the dorsolateral dislocation in the Bennett Fracture
- Carpal tunnel syndrome has an association with dialysis
- Degree of femoral head displacement predicts risk of AVN
- Proximal pole of the scaphoid is at risk for AVN with fracture
- Most common cause of sacral insufficiency fracture is osteoporosis in old lady
- Patella dislocation is nearly always lateral
- Tibial plateau fracture is way more common laterally
- SONK favors the medial knee (area of maximum weight bearing)
- Normal SI joints excludes Ank Spon
- Looser Zones are a type of insufficiency fracture
- T score of -2.5 marks osteoporosis
- First extensor compartment = de Quervains
- First and Second compartment = intersection syndrome
- Sixth extensor compartment = early RA
- Flexor pollicis longus goes through the carpal tunnel, flexor pollicis brevis does not
- The pisiform recess and radiocarpal joint normally communicate
- The periosteum is intact with both Perthes and ALPSA lesions. In a true bankart it is disrupted.
- Absent anterior/superior labrum, along with a thickened middle glenohumeral ligament is a Buford complex.
- Medial meniscus is thicker posterior.
- Anterior talofibular ligament is the most commonly torn ankle ligament
- TB in the spine - spares the disc space.
- Scoliosis curvature points away from the osteoid osteoma
- Osteochondroma is the only benign skeletal tumor associated with radiation.
- Mixed Connective Tissue Disease requires serology (Ribonucleoprotein) for Dx
- Medullary Bone Infarct will have fat in the middle
- Bucket Handle Meniscal tears are longitudinal tears

Section 12: mammo

Gamesmanship: “The calcifications don’t change configuration on CC and MLO views. This is the so called “tattoo sign” for dermal calcifications. Next step would be a tangential view to prove it.

Gamesmanship: Remember that secretory calcifications occur after menopause. Don’t call them secretory in a premenopausal patient (no matter how much they look like them).

Gamesmanship: If they show you a ML view for calcifications. Think hard about milk of calcium - is it tea cupping?

Gamesmanship: If a test writer wants you to say DCIS they can prompt it 3 ways: (1) suspicious calcifications (fine linear branching or fine pleomorphic), (2) non mass like enhancement on MRI, or (3) multiple intraductal masses on galactography.

Gamesmanship: Skin thickening and trabecular thickening should get progressively better with time. It should start out worst, then better, then better. If it gets worse - this recurrent disease.

Gamesmanship: Gynecomastia looks like a cancer on ultrasound. This is why a male breast cancer workup (palpable finding) always begins with a mammogram

When I Say This.....You Say That,

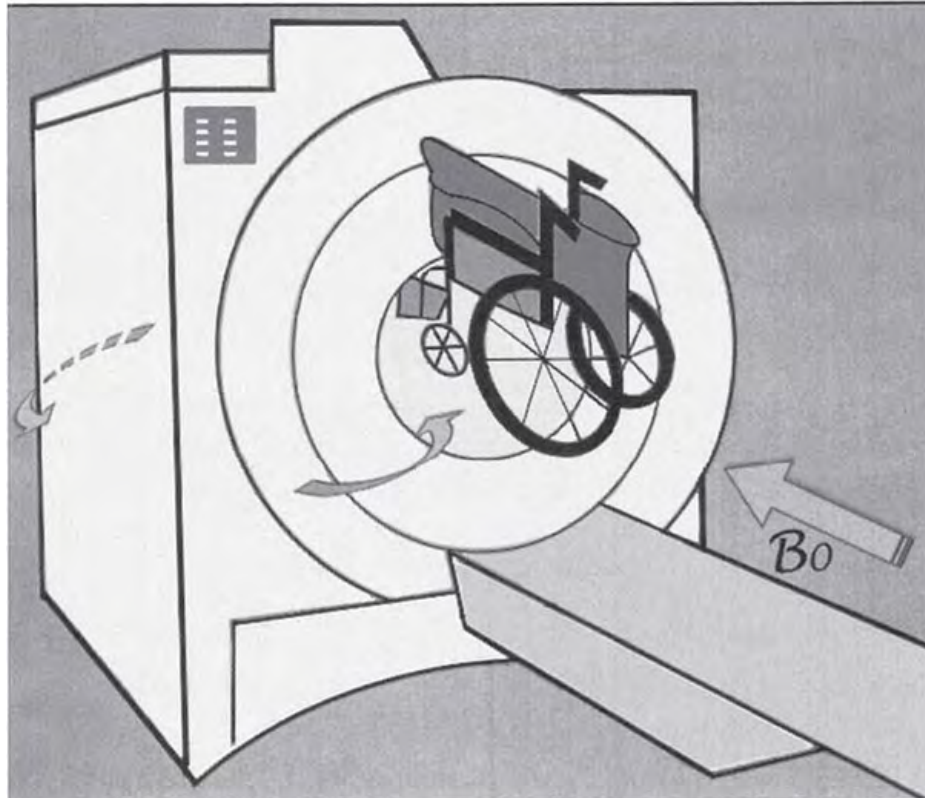

- When I say “shrinking breast,” you say ILC
- When I say “thick coopers ligaments,” you say edema
- When I say “thick fuzzy coopers ligaments - with normal skin,” you say blur
- When I say “dashes but no dots,” you say Secretory Calcifications
- When I say “cigar shaped calcifications,” you say Secretory Calcifications
- When I say “popcorn calcifications,” you say degenerated fibroadenoma
- When I say “breast within a breast,” you say hamartoma
- When I say “fat-fluid level,” you say galactoceles
- When I say “rapid growing fibroadenoma,” you say Phyllodes
- When I say “swollen red breast, not responding to antibiotics,” you say Inflammatory breast CA
- When I say “lines radiating to a single point,” you say Architectural distortion.
- When I say “Architectural distortion + Calcifications,” you say IDC + DCIS
- When I say “Architectural distortion without Calcifications,” you say ILC
- When I say “Stepladder Sign,” you say Intracapsular rupture on US
- When I say “Linguine Sign,” you say Intracapsular rupture on MRI
- When I say “Residual Calcs in the Lumpectomy Bed,” you say local recurrence
- When I say “No calcs in the core,” you say milk of calcium (requires polarized light to be seen).

High Yield Trivia

- No grid on mag views.
- BR -3 = < 2% chance of cancer
- BR-5 = > 95% chance of cancer
- Nipple enhancement can be normal on post contrast MRI - don't call it Pagets.
- Upper outer quadrant has the highest density of breast tissue, and therefore the most breast cancers.
- Majority of blood (60%) is via the internal mammary
- Majority of lymph (97%) is to the axilla
- The sternalis muscle can only be seen on CC view
- Most common location for ectopic breast tissue is in the axilla
- The follicular phase (day 7-14) is the best time to have a mammogram (and MRI).
- Breast Tenderness is max around day 27-30.
- Tyrer Cuzick is the most comprehensive risk model, but does not include breast density.
- If you had more than 20Gy of chest radiation as a child, you can get a screening MRI
- BRCA 2 (more than 1) is seen with male breast cancer
- BRCA 1 is more in younger patients, BRCA 2 is more in post menopausal
- BRCA 1 is more often a triple negative CA
- Use the LMO for kyphosis, pectus excavatum, and to avoid a pacemaker / line
- Use the ML to help catch milk of calcium layering
- Fine pleomorphic morphology to calcification has the highest suspicion for malignancy
- Intramammary lymph nodes are NOT in the fibroglandular tissue
- Surgical scars should get lighter, if they get denser - think about recurrent cancer.
- You CAN have isolated intracapsular rupture.
- You CAN NOT have isolated extra (it's always with intra).
- If you see silicone in a lymph node you need to recommend MRI to evaluate for intracapsular rupture
- The number one risk factor for implant rupture is the age of the implant
- Tamoxifen causes a decrease in parenchymal uptake, then a rebound.
- T2 Bright things - these are usually benign. Don't forget colloid cancer is T2 bright.

15 Physics

Prometheus Lionhart, M.D.



Physics for the CORE is supposedly much less math heavy than the old physics exam. The focus is more likely to be on practical problem solving, patient safety, and artifact reduction.

Things to know:

- Anything related to dose
- Artifacts

Section 1: X-Ray Production / Generation

What is Electromagnetic Radiation? This is basically a wave of energy that does not require a media to travel in (it can travel in a vacuum). Its velocity is fixed at 3×10^8 m/s

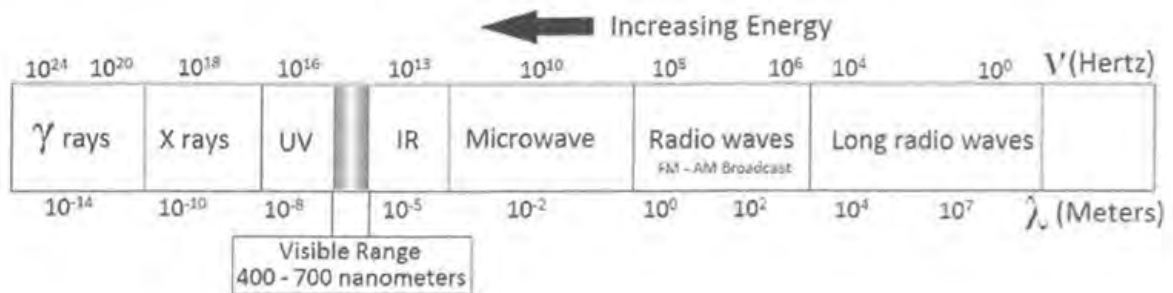
Remember these from general chemistry?

$$\text{Velocity} = \text{Frequency} \times \text{Wavelength}$$

$$\text{Energy} = \text{Frequency} \times \text{some constant "h"}$$

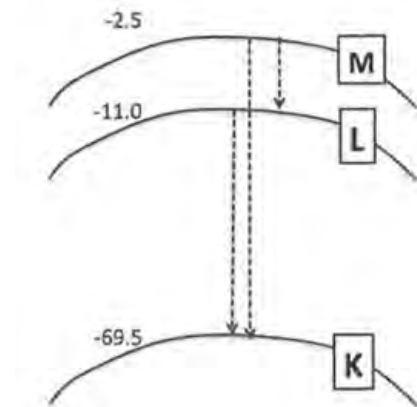
Because the velocity is fixed, frequency and wavelength have an inverse relationship. As one goes up, the other goes down.

Depending on the frequency and wavelength - an electromagnetic wave has different qualities. For example, long wavelengths (low frequency) can carry radiant heat. Whereas, shorter wavelengths (high frequency) can carry radio, tv, and radar signals.



As the x-ray frequency increases the wave gets more energetic and will have the ability to remove electrons from an atom. This is called **“ionizing radiation.”** Since it takes 15eV of energy to remove an electron from an atom, **a photon with energy greater than 15ev would be ionizing.**

Atomic Structure: Everyone knows electrons orbit the nucleus of protons and neutrons. Each one of these orbits has defined energy levels. **The inner most electron orbit is known as the K shell.** As the negatively charged electrons are attracted to the positively charged nucleus the K shell is the most desired location for electrons. It's not going to leave this desired spot unless energy is added to it. The electrons in higher orbits are in a "higher energy state" and require much less energy to remove. The outer most electrons require the least amount of energy to remove. Electrons can move between atomic shells, which requires the exchange of energy. Because an interior shell is a lower energy state, an electron from an outer shell will naturally "fall" closer to the nucleus to fill the vacancy.



When an outer electron moves to an inner shell, energy is released. If the energy is enough an x-ray may be produced. These x-rays are called "characteristic x-rays". More on these later.

X-Ray Production: X Rays are made by "*Thermionic Emissions of Electrons.*" This is done by taking a filament and running some current through it. The filament is typically made of Tungsten - because Tungsten has a very high melting temperature (it can tolerate a lot of heat). Sometimes rhenium (10%) is added to keep the Tungsten from cracking from heating and cooling. This current is considered "small", but it still makes the Tungsten really hot (over 2000 degrees) and therefore makes the electrons in the Tungsten atoms very energetic - until they "boil off."

The filament is a cathode (negatively charged). This repels the electrons as they boil off. They fire down the vacuum chamber towards a positively charged anode (also usually made of Tungsten).

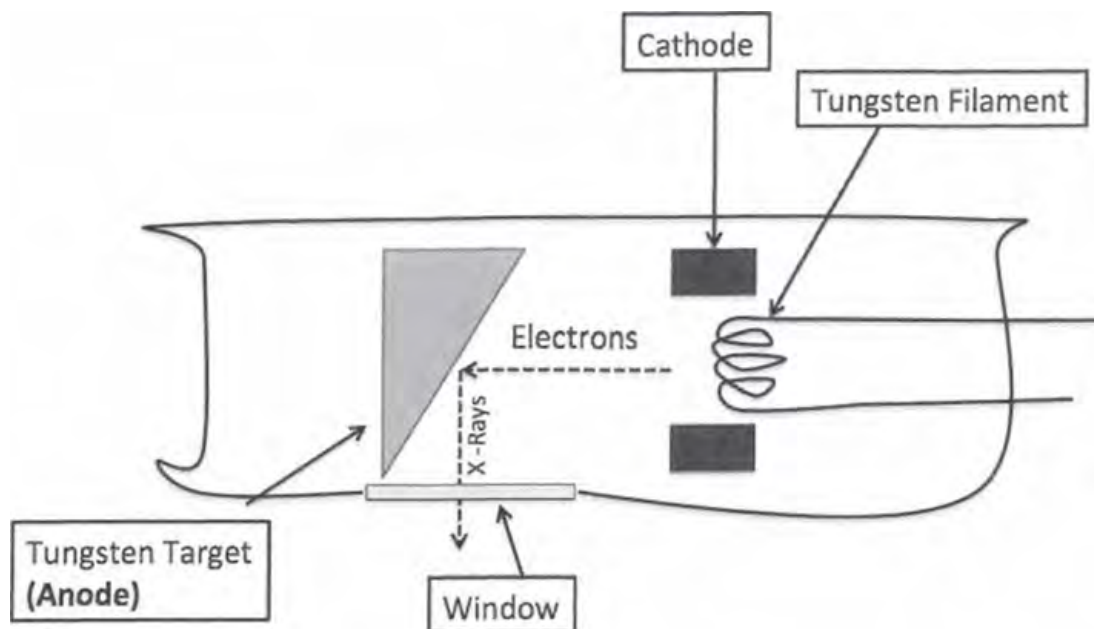
Why is the anode also made of Tungsten?

- Because it can tolerate a lot of heat.
- Because of its high Z , X ray yield is proportional to Z squared

Vocab:

- * *Space Charge Limited* - At low peak voltage, the potential is not enough to cause all the electrons to be pulled away from the filament, leaving a residual space charge remaining
- * *Saturation Voltage* - All electrons are immediately pulled away from the filament, and the tube current is maximized.
- * *Emission Limited* - Above 40 kVp, the filament current is proportional to and determines the tube current

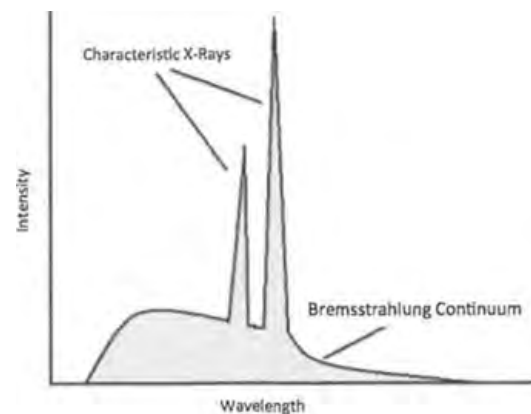
The free electrons accelerate toward this target (the anode) because of the potential difference between the cathode and anode. As they accelerate, they gain kinetic energy (keV). When the energetic electrons strike the tungsten target, they lose their kinetic energy via 3 different methods (excitation, ionization, and radiative losses i.e. Bremsstrahlung). X-rays are produced as described below.



Excitation - This occurs when you have energy transfer from the incident particle to an orbital electron in the target. The transfer moves the electron to a high energy state, but not high enough to actually remove it from the shell. When it finally calms down (returns to a lower energy state) **there is emission of heat - but no x-ray production.**

Ionization - If the energy of the incident particle is enough to eject an electron then you have ionization.

Characteristic Radiation (characteristic x-rays): When a vacancy in an inner shell is created, an electron in an outer shell promptly jumps in to fill the vacancy. Energy released in this process is equal to the difference in binding energies between the two shells. The energy may appear as an x-ray photon. Since the binding energies have exact characteristic values, the emitted x-rays carry exact and discrete energies and are called characteristic x-rays. The characteristic x-rays are depicted as two sharp peaks over the Bremsstrahlung continuum. The peaks noted on the figure correspond to the energy difference between the inner vacated shell (secondary to the inner shell electron being ejected upon collision with an incident high energy electron) and the outer shell which is vacated to fill the inner shell.

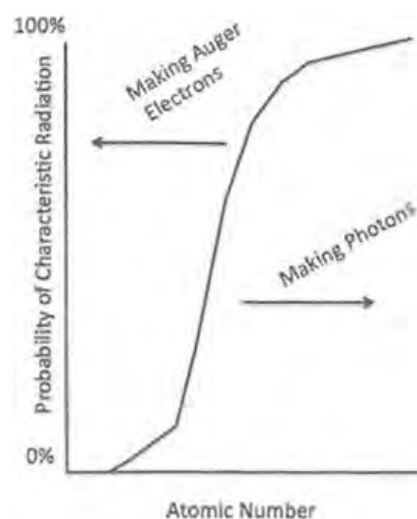


Testable Point: The K-shell binding energy of tungsten is -69.5 keV.

Testable Trivia: “No resultant (created) photon can have more energy than the incident electron”

Testable Trivia: K shell binding energy is proportional to Z^2 - so lower Z gives lower energy x-rays

Auger Electrons: If the energy released from the filling of an inner shell vacancy by an outer shell electron is imparted to another electron (instead of being emitted as a photon), which is then also ejected, the second ejected electron is referred to as an Auger electron. No x-rays are emitted in this process. In general, *heavy elements are likely to emit x-rays and lighter elements are more likely to emit Auger electrons.*



Secondary Ionization: Secondary ionization results when the electron which is ejected from the atom has enough kinetic energy to cause additional ionization events (i.e. eject additional electrons). The ejected electrons are sometimes called “delta rays.”

Bremsstrahlung (radiative losses) - Energized electrons are slowed down (braking) by the positively charged nucleus. As they slow down and approach the nucleus they have basically three responses. They can (1) strike the nucleus and give off maximum energy to the x-ray, (2) come close to the nucleus and give off medium energy to the x-ray, or (3) travel distant to the nucleus and give off a little energy to the x-ray. Most (80%) x-rays are produced this way.

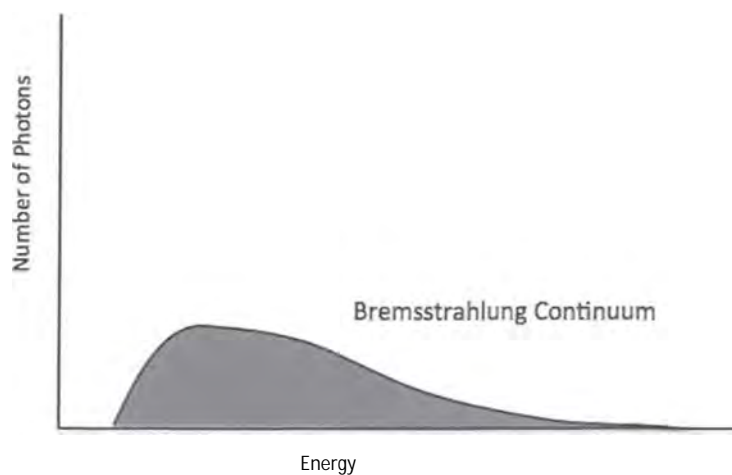
The amount of bremsstrahlung interactions is proportional to the energy of the incoming charged particle and the atomic number Z of the absorber.

Testable Trivia: Higher Z = More Bremsstrahlung

Testable Trivia: In nuclear medicine, low Z materials (plastic) are used to shield Beta emitters (classical example = ^{90}Y trium) in order to minimize bremsstrahlung production. Lead would make this much worse.

There are a range of energies produced. The critical point (highly testable) is that the maximum energy is equal to the max kVp.

Testable Trivia: Max energy = Max kVp.



There is a hump, because the low energy x-rays are immediately attenuated.

X-Ray Generator:

Acceleration of electrons from the cathode to the anode requires a large voltage. The characteristics of said voltage will contribute to the changes in the x-ray spectrum.

Three main factors are manipulated; kVp, mA, and the duration (time)

Factor	Trivia
mA	Controls the current through the cathode.
	Controls the amount of thermionic emission.
	Determines the <i>Quantity</i>
kVp	Controls the voltage between the anode and cathode
	Controls the kinetic energy given to electrons
	Determines the <i>Quality</i> of the spectrum
	Defines the maximum energy

mA vs kVp on intensity: X-ray production increases in a linear direction with mA. Doubling mA will double intensity of the x-ray spectrum. Increasing kVp by 15% will double the intensity of the spectrum.

mAs - This is the product of mA x expose time in seconds. You can think about this as the total amount of x-rays made and the total amount of heat made.

Heat Production Math: A general idea of how much “heat” is being produced may help you wiggle through some multiple choice questions.

$$\text{Tube Power} = \text{kV} \times \text{mA} \quad \text{Heat Units} = \text{kVp} \times \text{mA} \times \text{Seconds}$$

$$\text{Example} = 130 \text{ kv} \times 190 \text{ mA} = 24,700 \text{ watts or } 24,700 \text{ J for 1 sec exposure.}$$

*Remember a “Watt”
is a Joule per Second*

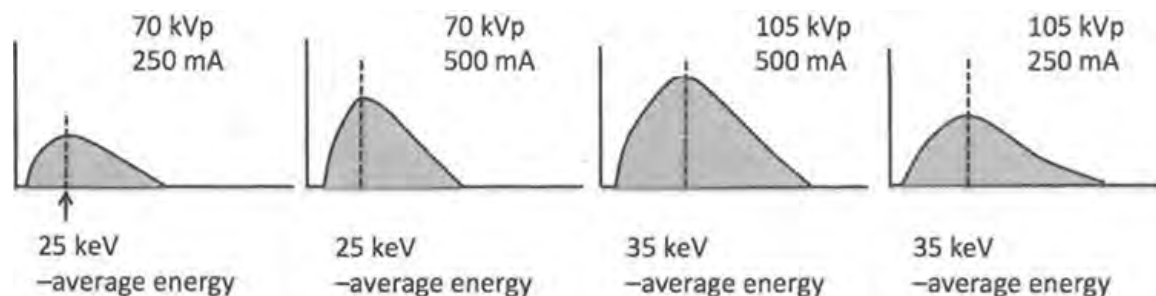
For regular diagnostic imaging, because your exposure times are so short you can have really high mA (like 1000). The limits of mA and kVp are based on tube heating and cooling specifics (now automatically controlled). Back in the stone ages, x-ray techs had to pick mA, kVp, and exposure time by guessing how fat someone was (sorta like one of those circus booths). They had charts on the wall to show max settings, otherwise they would melt the tube.

“Average Energy” - This is based on how much attenuation occurs at the target, as the x-ray exits the window, and as it’s collimated.

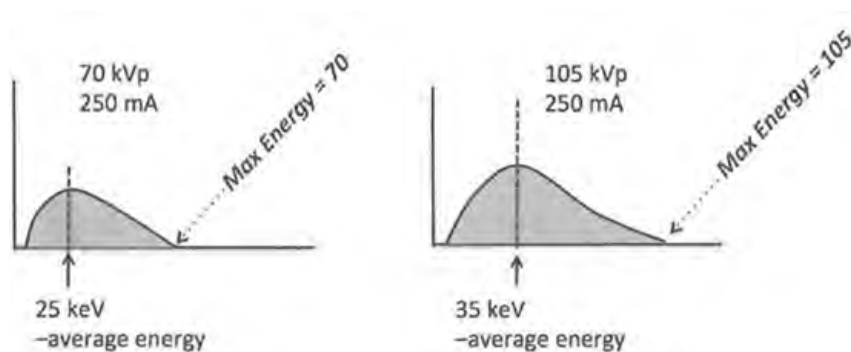
Testable Trivia: In a standard Tungsten target x-ray tube with normal filtration, the average energy is going to be between 1/3 to 1/2 the maximum energy.

X-Ray Spectrum Manipulation

All 4 of these are same target (Tungsten)

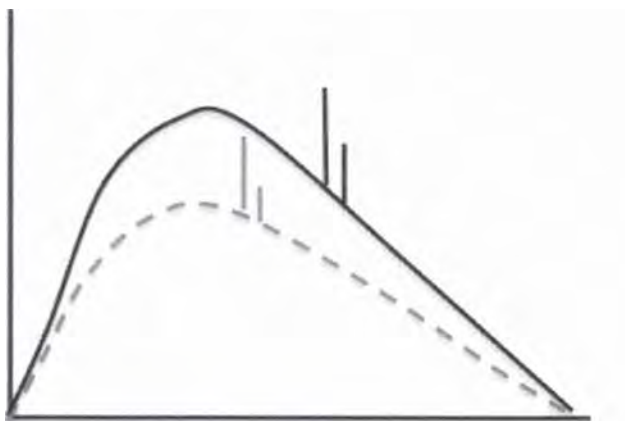


- * We are ignoring characteristic x-ray (they are all the same target)
- * Notice the change in the area under the curve with increasing mA
- * Notice the change in average energy with changing kVp



- * Notice the change in Max energy with changing kVp

Now let's look at some sneaky things you can do with the characteristic peaks.



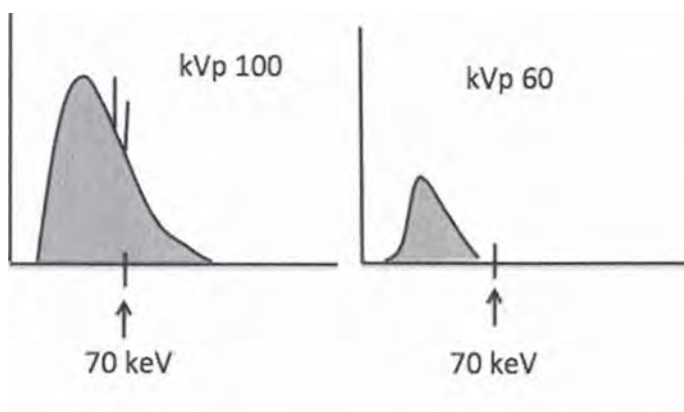
Two Superimposed Curves

Changing Targets

*Notice the characteristic peaks changed - this is how you know the target material is changed

Loss of Characteristic X-rays

*If you drop the kVp below the threshold for k shell electrons you are going to lose those characteristic peaks



Contrast and Dose:

Remember I mentioned that there is a “15% Rule of Thumb” - if you want to increase the x-ray exposure you can either (1) double your mAs or (2) increase the kVp by 15%.

Why you might do one vs the other depends on the need of the study. Don't forget that the average energy of the x-ray spectrum defines the attenuation characteristics and defines the contrast between objects and tissue.

Want to visualize low contrast objects / tissue ? - Keep the kVp constant and increase the mAs.

Want to lower dose but maintain a constant exposure ? Raise the kVp by 15%, then lower the mAs by 50%. *This works because the higher kVp x-rays will penetrate more easily and delivers a lower dose.

The study is contrasted ? You are going to want the kVp set at least twice the binding energy of the contrast agent being used. This maximizes your contrast (lets you see white on black).

- Iodine - K edge is 33 keV; so you want it set at least 66 kVp
- Barium - K edge is 37 kev, so you want it set at least 74 kVp

The Pediatric (Newborn) X-Ray:

When you x-ray an infant you are going to do a couple of things differently (which makes them testable).

- (1) You **do NOT use a Grid.**
- (2) You **lower the kVp** - “good technique is around 65 kVp.” They are small and don't require a lot of juice to penetrate. *Most adult CXRs are around 120-140 kVp.
- (3) You use around the same or lower mAs - “good technique is around 2-4 mAs.” Adults are typically around 4 mAs (portables are usually closer to 2.5).

Voltage Transformer:

Voltage is what gets the electrons accelerated to the anode. Moving these electrons actually requires a lot of voltage, and because AC current is used it needs to be stepped up through a transformer to the kilovolt range. The problem with alternating current is that it alternates. This essentially turns the tube on and off - no good. This was fixed by removing the voltage in the wrong direction with a circuit called a "*rectifier circuit*."

"*Voltage Ripple*" - Older generators just turned the current with a "rectifier circuit". This voltage varied a lot (zero to max), causing "ripples." An uneven x-ray voltage is not efficient. The most efficient tube voltage is constant at maximum value. This was fixed by engineering multiple single phase circuits or a higher frequency inverter. Modern generators are either three phase or have higher inverters - so voltage ripple just doesn't happen anymore. So, because this problem has been fixed and you will never encounter it in your entire career - it's probably high yield.

The Focal Spot:

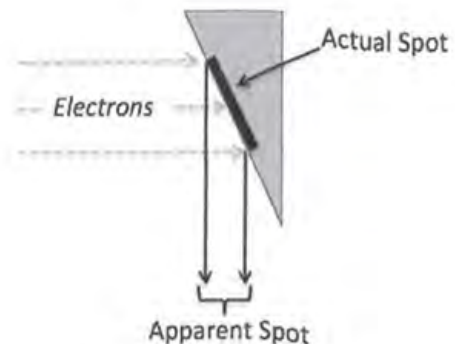
The focal spot is the area of electron bombardment (and x-ray production) on the anode. Generally speaking a smaller anode has better spatial resolution. The trade off is that a smaller anode can't disperse heat as well and can melt (so you deal with more heat limitation). This is addressed in two main ways: (1) angling the anode - gives a larger surface area, (2) using a rotating anode.

Testable Trivia:

- Mammography uses a focal spot of 0.3 and 0.1 mm
- General X-ray uses a focal spot of 0.6 and 1.2 mm
- Portable x-ray device often use a stationary anode (doesn't rotate to dissipate heat). This limits their tube rating.

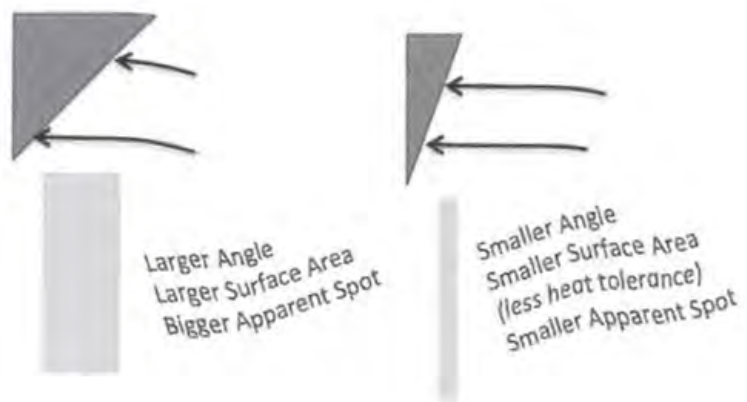
Vocab:

- *Actual Focal Spot*: This is where the x-rays land on the target anode.
- *Apparent Focal Spot*: This is where the x-rays land on the patient. This **defines the amount of blur** (not the actual spot).
- "*Line Focus Principle*" - This is the method of angling the anode to give you a smaller apparent focal spot (less blur).



Changes in Angle:

The goal is the smallest focal spot possible (best resolution) - the trade off is heat tolerance.



The Effect of mA/ kVp on Focal Spot

The actual focal spot will enlarge with an increase in mA. The reason is, you have more electrons and they start to repel each other “*elbow each other out of the way*” This results in “*blooming*” or widening of the beam. This repulsion is most significant at low kVs. With increased kV you can get a slight decrease or “*thinning*” of the focal spot.

High mA, Low kVp = Wider Spot “blooming”

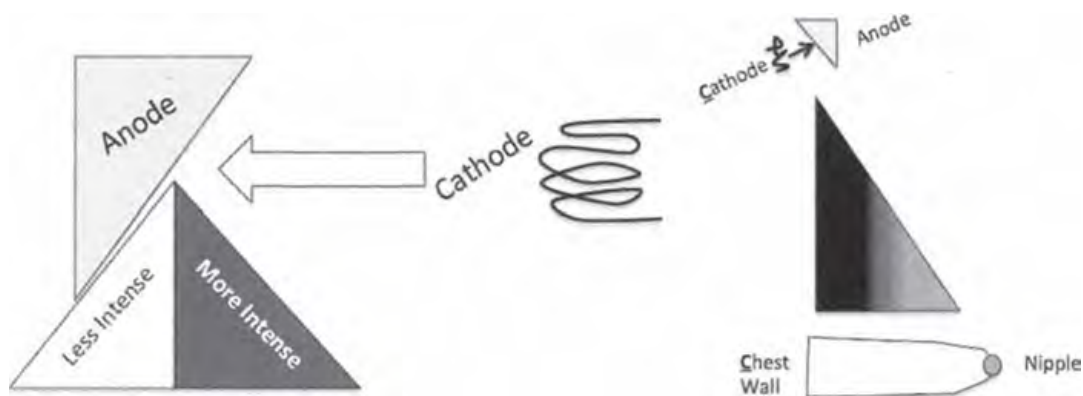
High kVp = Smaller Spot “thinning”

The Heel Effect:

Because x-rays on the anode side must pass through a greater thickness of the anode, you have a reduction in the intensity of these x-rays.

Testable Trivia: The heel effect can be decreased by increasing the anode angle or decreasing the size of the x-ray field.

Testable Trivia: This is used in mammo, where the thicker part of the breast / chest wall are aligned with the cathode (“*Cathode on the “Chest wall*”).



Magnitude of the Heel Effect

Depends on 3 Factors

- (1) Anode Angle - *Worse with Small Angle*
- (2) Source to Image Distance - *Worse with Decreased SID*
- (3) Field Size - *Worse with Bigger Field*

Heel Effect on Field of View: Because the energy decreases on the anode side, this also decreases your field of view on that side (the side of the nipple). To compensate for this the entire tube is angled up to 20 degrees.

“Effective Anode Angle” - This is the sum of the anode and tube angles.

Understanding How the Anode Angle Changes the Heel Effect:

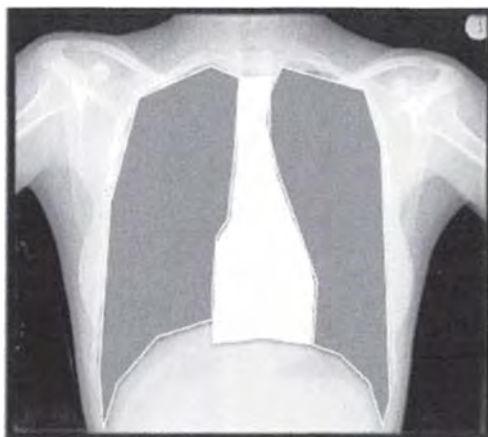
The sharper the angle, the more abrupt the change in intensity and therefore the more heel effect. I like to think about this as the “heel cut off.” As, you get a sharp transition with a small angle, and a more gradual change with a larger angle.

Smaller Angle = Greater Heel Effect (heel cut off)

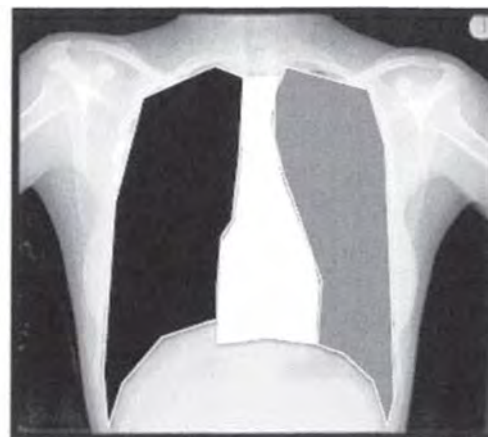


Another way to show the Heel Effect:

The classic way to show the heel effect is with a mammogram (cathode on the chest wall). However, there is another sneaky way to show this principal and that is with a chest x-ray.



Proper Technique:
-Anode - Cathode
Oriented Vertically
With the Cathode Down



Wrong Technique:
-Anode - Cathode
Oriented Horizontal
Creates a Unilateral
Lucent Lung



Misc Topics:

Off-Focal Radiation: This is scatter from the anode outside the focal area. This leads to an *increase in patient exposure and blurring*. This can be reduced with a small lead collimator near the tube output. Tubes constructed with metal enclosures and/or with the anode at electric ground potential will have less off -focal radiation (metal envelope attracts the scattered electrons).

X-Ray Tube Insert: X-ray tube is sealed under high vacuum. This vacuum is needed to prevent electrons from interacting with gas molecules. The x-ray port is necessary because x-rays are produced in all directions. Only the x-rays heading towards the patient are “useful.” The port is typically made of the same stuff as the housing (except with mammography in where it’s made of beryllium to minimize low energy x-ray absorption).

Unwanted Radiation Vocab:

- **Leakage** - X-rays that are transmitted through the housing
- **Secondary** - Characteristic radiation that is made from electron interaction with materials other than the target (glass, housing, etc.).
- **Scattered** - X rays that are deflected in direction once they leave the tube
- **Stray** - The sum total of leakage and scatter

Collimation: This is the process of restricting the size and shape of the x-ray beam emerging from the port. It’s done to reduce both primary and secondary radiation. It improves image quality (less fog). The tech can gauge how much they are collimating by using a beam of light reflected by a mirror of low x-ray attenuation that mimics the x-ray beam. By order of various regulatory bodies, the light field and x-ray field must closely align.

Grid Controlled: This has nothing to do with an actual grid. Instead it refers to a way to turn the tube on and off fast by using a little cup around the filament to “suck electrons out.” You are not actually shifting the big voltage across the tube, just diverting the electrons. This is how *pulse fluoro* is done.

Half Value Layer (HVL):

This is a measurement that is done to help understand beam quality. It is the amount of material required to attenuate an x-ray to 1/2 the original output. The higher the average photon energy the more penetrating it will be, and the larger its HVL.

Beam Filtration: If the beam is filtered the whimpy low energy photons will be removed first (leaving the higher ones). This will increase the average photon energy. Since **average photon energy determines penetration capacity** this will increase the HVL.

• **More filtration = Higher HVL**

• **Less filtration = Lower HVL**

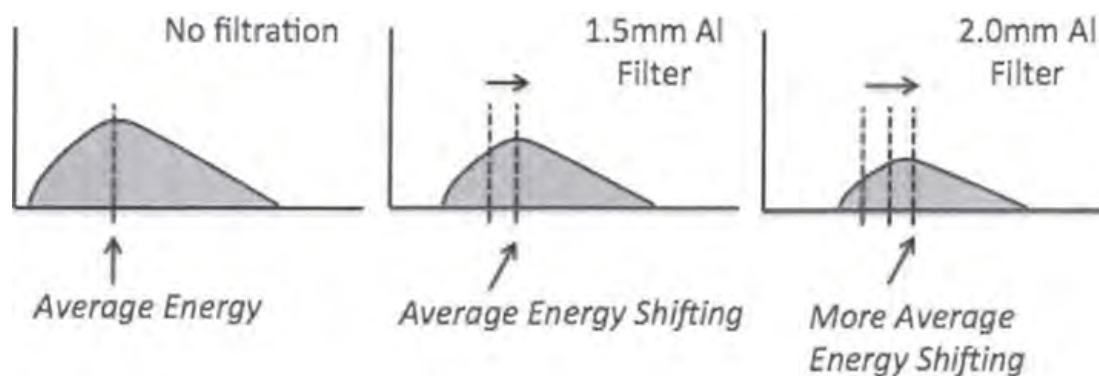
Testable Trivia: With each HVL the average photon energy goes up.

3rd HVL > 2nd HVL > 1st HVL

Key points:

- HVL of an xray beam does NOT depend on mAs
- HVL does depend on beam filtration
- HVL does depend on anode material (e.g. tungsten)

Lets look at some sneaky ways this can get graphed out:



• Notice the average energy increases with more filtration

• Notice the area under the curve is decreasing as well (you are losing x-rays).

Testable Trivia: A mono-energetic beam would have a higher HVL than a poly-energetic beam (at the same kVp).

10th HVL (“TVL”)

This is the thickness of material that can attenuate an x-ray to 90%. This is used for shielding calculations.

“If you will it, it is no dream ” - Theodor Herzl

Section 2: X-Ray Interaction with Matter

Interactions with “matter,” really means interactions with “people.” Several types of interactions that can take place, but... but from a clinical standpoint only three matter: Coherent, Compton, and Photoelectric.

Coherent Scattering (Rayleigh Scattering): This occurs when a photon excites the entire atom. Eventually you get de-excitation and a photon is emitted (same energy but different direction as the original photon). If the photons produced with this kind of scatter reach the image receptor they can cause some loss in contrast.

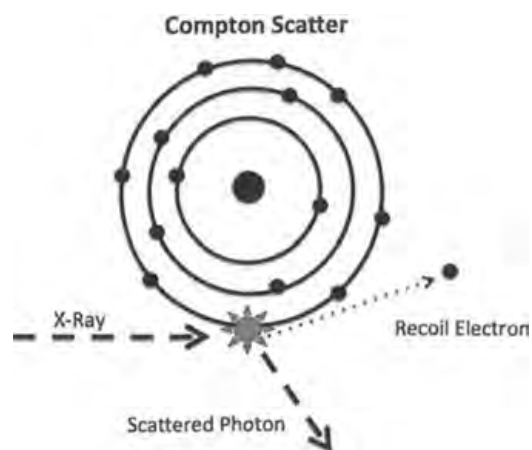
Key Points:

- This (*Coherent Scattering*) does NOT result in ionization (no electron is lost).
- This does NOT result in the net transfer of energy.
- This does NOT result in any dose to the patient.
- This does NOT generate an X-ray
- Seen primarily at low energies - i.e. mammography ; wasting about 15% of photon interaction below 30 keV.

Compton Scattering (the had guy): This occurs when an x-ray hits an *outer shell* electron. The energy from this x-ray is transferred to the electron, which is then ejected. The incoming x-ray/photon (now with slightly less energy) changes direction and flies off. The amount of energy transferred has to do with the angle at which it strikes the electron (direct hit more energy, glancing blow less energy).

Compton Scattering produces 3 things:

- (1) Free Electron
- (2) Ionized Atom (missing an electron)
- (3) Photon of energy

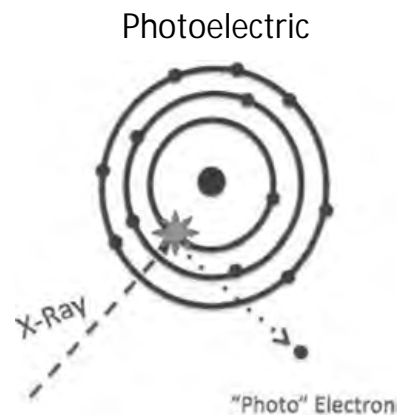


Key Point:

- Probability of a Compton scatter does NOT depend of the Z of the atom - because the energy of these outer shell electrons is low.
- Probability of a Compton scatter is dependent on the density of the material - more tightly packed atoms, means more electrons to crash into
- Above 25-30 keV Compton scatter is the dominant photon interaction in soft tissue

Photoelectric Effect (the good guy): This occurs when an x-ray hits an inner shell electron. If the energy is great enough to overcome the k-shell binding energy then the inner shell electron will get ejected -this is an *all or nothing* reaction. The ejected electron is called the “photo electron. ”

With this inner electron spot missing you get downward cascade and release of a characteristic x-ray or you get the production of an Auger electron. Because Auger electron production tends to dominate in biologic tissue (unlike Tungsten, a the target with a high Z) you still end up with biologic damage from these free electrons. P.E. is still good because this actually contributes to image contrast (Compton just makes noise).

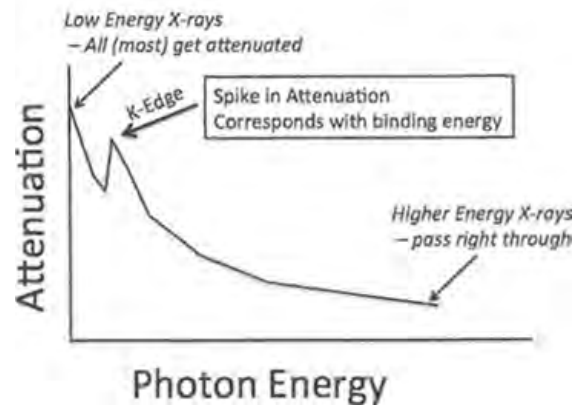


Key Points:

- Probability of P.E. directly proportional to the atomic number cubed.
- Probability of P.E. is increased with object density
- Probability of P.E. is inversely proportional to the energy cubed

Compton (Bad Guy)	Photoelectric (Good Guy)
Outer Shell Electron	Inner Shell Electron
Variable Energy Transfer	All or None
Does not care about Z	Depends on Z^3
Depends on Density	
Dominates above 30 keV	Dominates below 30 keV

K Edge: The chance of the photoelectric effect happening sharply increases when the photon energy and electron binding energy are the same. This is taken advantage of in imaging by using iodine (33 keV) and barium (37 keV) - which are right in the diagnostic range (when the kVp is set between 65 -90). The result is that iodine and barium will really soak up those x-rays, because of their K-edges, resulting in high contrast.



X-Ray Attenuation in Tissue:

Depends on 3 things:

- Effective Atomic Number in Tissue
- X Ray Beam Quality (energy)
- Tissue Density

Linear Attenuation vs Mass Attenuation:

- **Linear:** This is the actual fraction of photons interacting per unit thickness of an absorber. In other words, it is the fraction of photons removed from the x-ray in a certain distance factoring in effects from compton scatter, PE effect, and coherent scatter. In contrast to “mass attenuation,” the linear attenuation of ice, water, and water vapor is different (they have different lengths for the same amount of molecules).

Factors to consider

- * More attenuation occurs with denser object
 - * More attenuation occurs with higher Z material
 - * Lower attenuation occurs with higher kVp
 - * Higher attenuation occurs at K-edge
- **Mass:** This is the fraction of photons interacting - scaled per gram of tissue. This is *supposed to reflect the attenuation*. The important point is that the mass attenuation of ice, water, and water vapor is the same.

Is this your homework, Larry?

Section 3: General X-Ray Concepts

Image Noise - This is unwanted variation in image density. Noise varies from multiple factors including the thickness of film used. Quantum mottle obviously plays a role in general background graininess (*yes that's a word*).

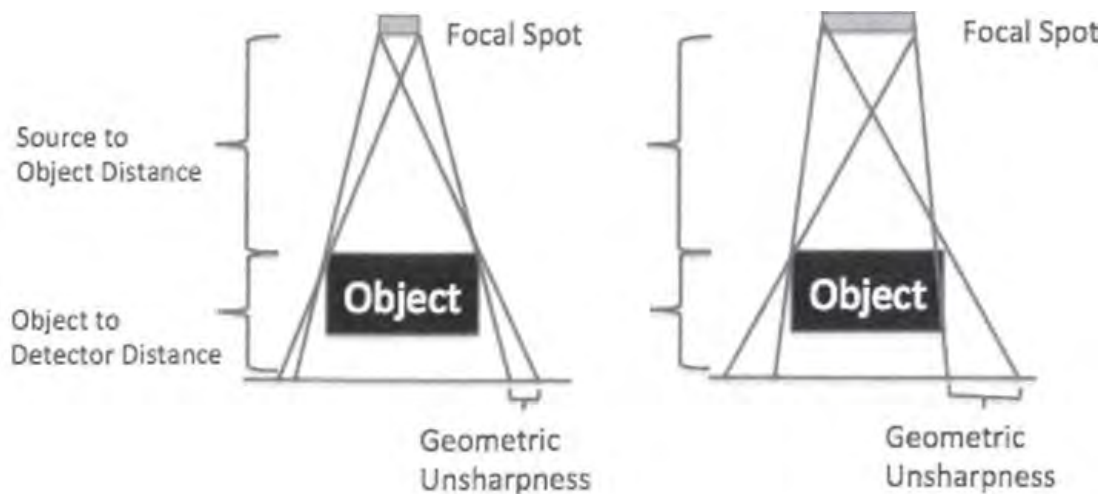
Testable Trivia: Noise will increase as the distance between the tube and detector increase, with the increase in noise described by the inverse square law.

Geometric Relationship - Influences Performance:

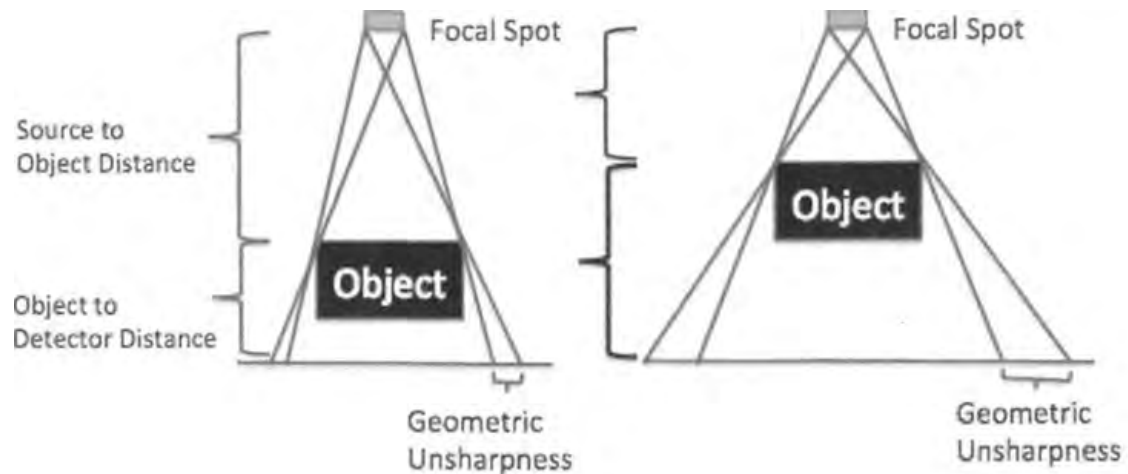
Geometric Unsharpness: This occurs because of several factors:

- Focal Spot: Small Focal Spot = Less Blur
- Source-Object Distance: Closer the source is to the image = More Blur
- Object Detector Distance: Closer the object is to the detector = Less Blur
- Magnification: **More Magnification = More Blur**

Below is a diagram showing how a change in focal spot will change the size of geometric unsharpness. As you can see, a smaller focal spot has less “unsharpness.”



Now let's look at some different distances. If you are asked about "source to object", or "object to detector" distances I recommend you draw them out. If you can draw it, it will save some room in your brain for memorizing stuff that really matters (like the dose for temporary epilation, or how often you do the "flood test").

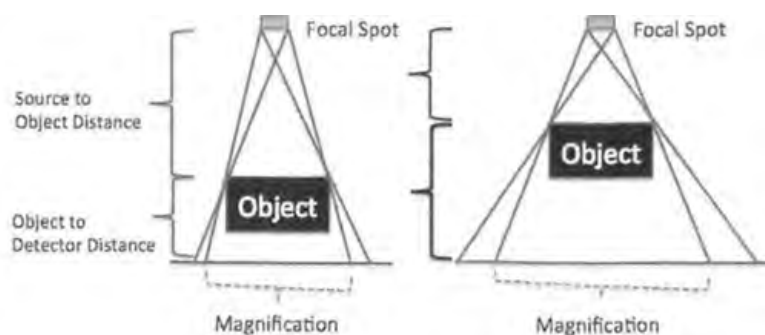


Magnification:

Let's take another look at that diagram. Notice the magnification difference between when you change the source to object distance?

Magnification is calculated by:

The tube to patient distance + patient to detector distance / tube to patient distance.



Magnification:

Increases with:

The patient far away from the detector.
"Greater Object to Detector Distance"

The source closer to the patient. **"Less Source to Object Distance"** *

**The Patient = Object*

****More on Magnification in the Fluoro Chapter***

Scatter:

They say scatter is a “violation” of the most basic law of radiography, because it doesn’t originate in a straight line from the focal spot. Instead it is the **result of compton scatter in the body.**

Scattering depends on:

- (1) The collimated field - *less field = less scatter*
- (2) Thickness of the imaged body part
- *thinner part = less scatter*
- (3) The energy that is chosen - *compton dominates above 26 kVp in soft tissue, and above 35 kVp in bone.*

Scatter

- Collimation
- “Thickness” of Patient
- Energy of the Beam

Reducing Scatter:

Grid - You can use a grid. Grids have different ratios. The more strips the higher the ratio and the more scatter reduction. If you are using a “linear” grid it needs to align parallel with the anode-cathode, to avoid “grid cut-off.” Cross-hatched or “cellular” grids don’t have this issue.

“Bucky Grids” - This is a moving grid. They wiggle back and forth rapidly - too fast to be seen. If the grid motion fails (and it stops moving) you will end up with grid lines.

“Bucky Factor” - The bucky factor describes the increased mAs required when a particular grid is used compared with a study using no grid.

Air Gap - This is basically only done with mag view on mammo. The idea is that the primary x-rays will shoot straight and go further than the scatter ones. So, if you create a little distance between the patient and the detector you can reduce the scattered photons actually being registered.

“Screwing up the Grid” - You can put the grid on upside down, off centered, or skewed. The chance of this happening is way more with a portable.

What does the Grid do to Dose ?: The **tradeoff to using a grid is that dose is increased** (because the automatic brightness control turns up the juice to compensate). There is a thing called the “bucky factor” which is basically the dose with the grid / dose without the grid. The most common bucky factor is around 2-3.

The Grid	
Pros	Cons
Reduces Scatter (improves contrast)	Increased Dose
	Increased Exposure time (possible motion artifact)

Digital vs Analog

Long ago, when dinosaurs roamed the earth, radiology was done on film screen. Now in the modern age it is digital. The comparison of film screen vs digital radiology could lend itself to multiple choice questions.

Film Screen Limitations: Screen systems are intolerant to errors in exposure (under or over leads to a loss of contrast). Noise - both on film and fluoro is an issue, with Compton scatter increasing with increasing film size. The quality of films breaks down over time. Film can't be manipulated post processing.

Possible Film Screen Advantage: **Spatial resolution of film screen is slightly better** than CR (5 line pairs per mm, vs 2.5 line pair per mm).

Digital Radiography Advantages: Digital images can easily be stored, and manipulated post processing. They don't break down chemically. **There is a higher dose efficiency (potential for less radiation) and wider dynamic range of detection.** Although the spatial resolution of film screen is slightly better, the superior contrast resolution of digital radiography more than compensates.

Digital: If you are working anywhere in the United States this is all you will use. If you are working in certain parts of Africa or have traveled back in time about 20 years you may still be using film.

Digital Pixels: Most systems use pixels with 8 bits, so this is 2^8 or 256 possible brightness values. Why did I use "2"? - it's a binary thing - black and white.

Digital detectors can be broken down like this:

- Storage Phosphor (CR) - Type of Indirect
- Flat Panel Detectors (DR)
 - Direct
 - Indirect

Computed Radiography (CR)	Digital Radiography
Uses a photostimulable phosphor plate enclosed in a cassette. Has a two stage process for image capture and image readout (done separately)	Uses a detector that can capture and read out information. Further classified into direct and indirect methods.

Storage Phosphors (CR) : These can work with a conventional x-ray machine (just like a normal cassette). The system uses a special phosphor (*Barium Fluorohalides*) that does not emit the absorbed energy as light after interaction with an x-ray. Instead, the x-ray causes an electron in the phosphor to change to a metastable state (one that it can hold for several days).

The idea is that the storage phosphor is holding a “latent” x-ray image. The information is read by using a red point laser to scan the detector and count how much **blue-green** light is emitted as the high energy electron gets knocked out of it’s metastable state.

Testable Trivia: The amount of light detected is proportional to the intensity of the incident x-ray.

The photostimulated phosphors have a wide detectable range, tolerating x-ray intensities 100 times higher and 100 times lower than the 5 micro Gy needed for an old school screen film.

The plate is reset by forceable exposure to bright white light - which erases it. If you forget to do this you will get ghosting artifacts.

Spatial Resolution of CR - depends on:
Laser Spot Size
Phosphor Plate Density and Thickness
Rate of light sampling

Flat Panel Detectors (DR): When most people say “digital detector,” this is what they are talking about. It’s much faster than conventional film development or CR plate reading. These are composed of amorphous (not crystalline) selenium. The photon from the x-ray is stored as an electric charge within a square array of pixels. The information is read out by scanning the information a row at a time with all columns read in parallel.

What is the difference between direct and indirect?

- Indirect (scintillators) = X rays —>• Light —>• Charge
- Direct (photoconductors) = X rays — Charge

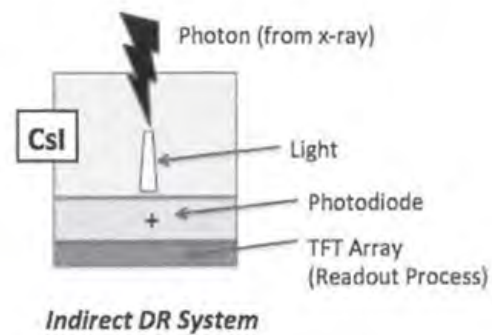
This vs That - Scintillators vs Phosphor

Scintillators usually have 2-3 times the efficiency in x-ray absorption of a Phosphor - at the same thickness.

Scintillators produce more visible light per x-ray

Scintillators emit a wavelength of light that is a better match for the a TFT detector

How does an indirect FPD take an x-ray and make it a picture? The x-ray activates the thallium doped Cesium Iodide (CsI), which emits light. The photodiode turns the light into an electric signal which can be read out.



What is this “Lateral Dispersion ” ? This is a problem you run into with particular phosphors. Light tends to diffuse laterally after it leaves the site of conversion from an x-ray. This creates issues with spatial resolution, that get worse with increasing thickness of the crystal. Making the crystal thin has its own problems as your sensitivity for collecting x-rays is going to drop off (they just fly right through).

Is there a difference in “Lateral Dispersion ” between phosphors? Yes - Gadolinium Oxysulfide has more lateral dispersion (light scatter) than CsI.

How does one solve the problem of “Lateral Dispersion ” ? Luckily someone much smarter than you or me already did. They invented a columnar structure that forms a “lead pipe” like matrix. With this design you can make a nice thick crystal without worry.

What is this “Thin-Film Transistor” (TFT) ? These are active electronic elements that can be used in both direct and indirect systems. It’s basically a layer of elements typically starting with readout electrons at the bottom and charge collector arrays on the top.

How does a direct FPD take an x-ray and make it “charge”, without first making it “light”?
- It uses a magical substance called amorphous selenium.

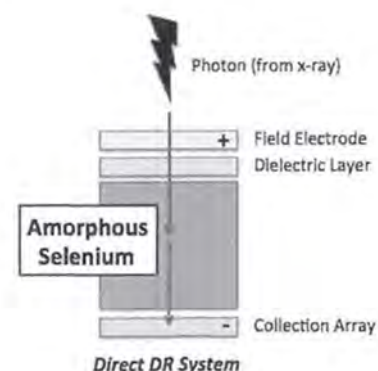
Step 1: Apply a homogeneous “bias” charge to the surface of the selenium.

Step 2: Fire the x-rays through the patient, into the selenium. The x-rays are absorbed by the selenium and electrons are released.

Step 3: Released electrons (“electron hole pair”) travel to the surface of the selenium and neutralize a portion of the applied charge. This is done in proportion to the radiation intensity. An important testable point is there is **no lateral dispersion**.

Step 4: These electrical charges are drawn in along the electric field lines to the charge storage capacitor electrodes connected to the TFT.

Step 5: The pattern of charges is scanned and converted to a digital signal stored by each TFT.



Amorphous Selenium

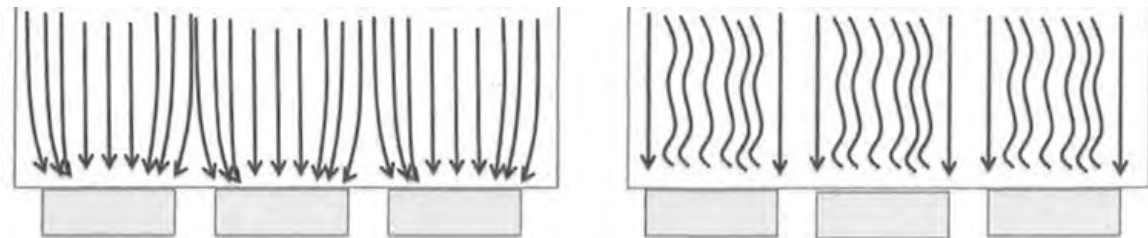
The material is magical, likely originating from the underground dwarf mines of middle earth. Selenium is essentially a photoconductor. When it is exposed to radiation, its electrical conductivity is altered in proportion to the intensity of the radiation.

It is very susceptible to humidity and temperature. Don't try and use it outside in the Amazon jungle.

What is this "Fill Factor" ? This is the area of the detector which is sensitive to x-rays (in relation to the entire detector area). The higher the fill factor, the more efficient the detector.

Fill Factor Differences:

With DR systems, the electric field shaping essentially allows for a fill factor of nearly 100%. This is not seen with Indirect (CR) systems. Remember, differences between things make good multiple choice questions.



Direct - Amorphous Selenium

Indirect - Cesium Iodide

Spatial Resolution: In the digital world the term "spatial frequency" is often used, which is good because it helps me remember that the more frequently an object is sampled the better the resolution.

Some General Pearls /Points (possibly testable):

- Increased sampling frequency = Increased Resolution
- More pixels = Higher spatial resolution
- Images with high resolution need large file sizes
- Increasing x-rays will NOT improve Maximum spatial resolution
- Spatial resolution for Selenium based (direct) DR is higher than indirect detectors *newer systems are pretty close actually
- Structured scintillators are better than unstructured ones (less lateral dispersion)

What is this “Modular Transfer Function?” (MTF)

Buzzword = Contrast.

Buzzword = “Function of Spatial Resolution (Frequency)”

Objects with different sizes and different densities are recorded at different gray scale values. The easiest way to think of “MTF” is that it is a method to describe the displaying of object contrast and size. It is the ability to take the contrast values (object contrast) and turn it into intensity levels in an image (image contrast).

Another way to think about MTF is a ratio of input and output. The fraction of signal contrast that will be maintained in the captured image.

MTF changes as a function of spatial frequency (resolution) - 100% at low spatial resolution, to zero at high spatial resolution.



What is this “Detective Quantum Efficiency?” (DQE) This is efficiency of a detector in converting x-ray energy into an image signal. People who like math calculate this by comparing the signal to noise ratio at output with the signal to noise ratio at input as a function of spatial frequency. In a perfect world you want this to be “1.0” (all radiation energy is absorbed and converted to image). Factors that influence it are the radiation exposure, spatial frequency (resolution), modular transfer function, detector material, kVp, and mAs. The better your DQE the less radiation you need to maintain your signal. Just like MTF, DQE is better at low spatial resolution. In general the DQE of DR is around 0.45, significantly better than that of CR or plain films - which is around 0.25.

Radiation Exposure: If you have fewer repeat exams (because you can actually do post processing) then you have less radiation - obvious advantage of digital radiography. Additionally, because DR systems have superior quantum efficiency, you can lower dose compared to CR or plain films and get the same quality. DR has less dose.

Screen Films: It’s true that board exams tend to lag behind reality for several years (sometimes decades), but I’ve put a lot of thought into this, and I refuse to include a complete section on screen films in this book. I don’t even know if they will actually have questions on screen films (*how much acetic acid do you need in your fixing solution?*), but if they do... well then just miss them.

Seriously, this has gone to far. It’s over the line. This isn’t Nam, there are rules.

OK fine, in the mammo chapter I'll talk a little about the film screen and lateral light diffusion, but that's it! I mean it... I'm not gonna say anything else about plain films.

Just know this: Screen films have a higher spatial resolution (sometimes) than digital systems.

Direct Conversion	Indirect Conversion
Directly converts x-rays to electrical signal	X-Ray -> Light -> Electrical Signal
Detector material is amorphous selenium	Phosphor material is usually thallium doped cesium iodine
Signal does not "laterally disperse", as the applied voltage separates the electrons and holes made by x-rays	Light can scatter (worse with thicker crystal), better if columnar structure is used.
Fill Factor is high (near 100%)	Moderate fill factor (depends on size of pixel)
Higher Detector Quantum Efficiency (DQE)	Moderate Detector Quantum Efficiency (DQE)

Film Artifacts

Fogging - This is the adding of charge to the detector (a blackening of the film). This can happen if you leave the cassette in the room with scattered x-rays. You can get a big black blob on it.

Double Exposure: This happens when the cassette is used twice without changing the film or erasing the receptor. The film will look like it has two images on it.

Quantum Mottle: This is noise from a lack of photons. The film will look underexposed.

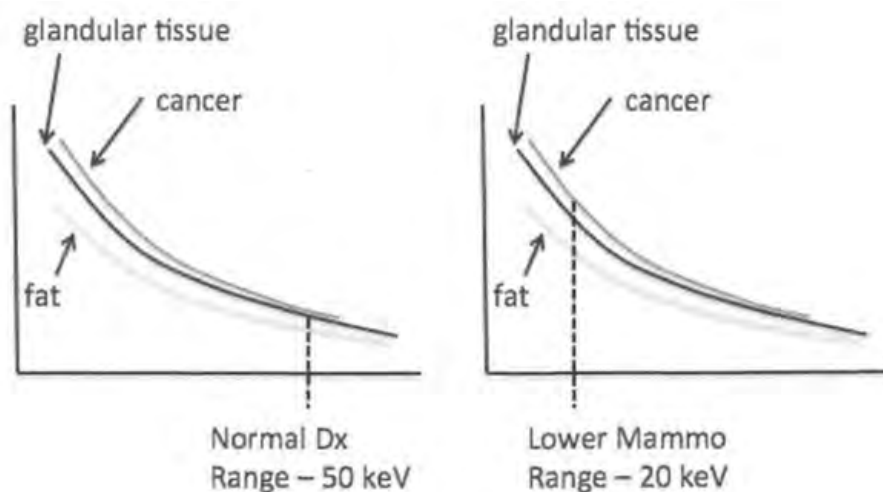
Incomplete Erasure: This looks like a double exposure.

Ghosting: This is the result of prior exposure leading to difference in x-ray sensitivity of different parts of the detector. This looks like a dark object that doesn't belong on the image.
The testable point is that this occurs in DR more than CR.

We're talking about unchecked aggression here, dude.

Section 4: Mammo

Why is mammo different than regular x-ray? The difference in attenuation of a breast cancer and just regular breast tissue is very small. So you have to use lower energy and a nearly mono-energetic beam to enhance the attenuation differences. Also, an increase in spatial resolution is required to see micro calcifications.

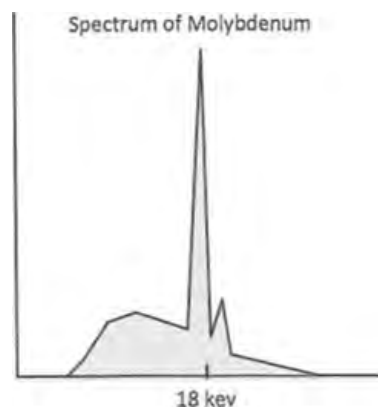


Optimal kVp: The ideal energy for mammo is between 16-23 keV. To get this energy a voltage of 25-30 kVp is used (general Dx uses between 50-120).

Target Anodes: Mammo uses a molybdenum or rhodium anode (general radiology uses tungsten).

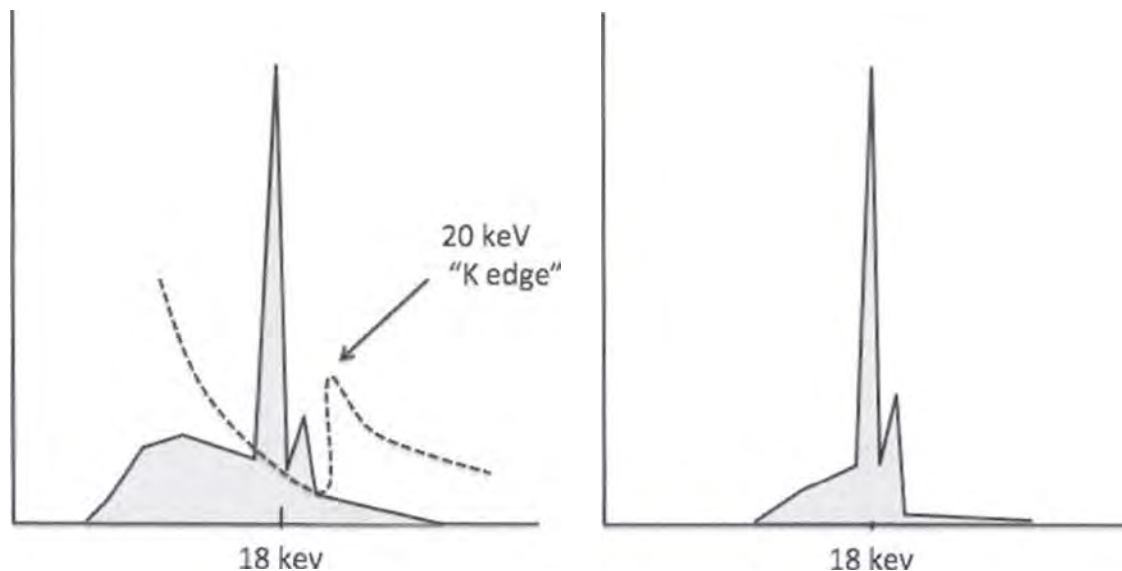
The Normal Spectrum of Molybdenum:

The combination of low kVp and a low Z give Moly high characteristic x-rays, with low Bremsstrahlung.



K Edge Filtration - A K edge filter is placed on the outside of the tube with the goal of creating a nearly mono-energetic beam in the target range of 16-23 keV. Moly is classically used for this, taking advantage of the 20 keV K edge and subsequent rise in photoelectric absorption. Energies lower than 15 and greater the 20 get filtered out.

Spectrum of Molybdenum with Superimposed Mo Filter



Rhodium: An alternative to Moly is Rhodium, which has a similar (slightly stronger) spectrum, and a binding energy of 20.2 keV.

High Yield:

- Filters block BOTH high and low photons, with the benefit of removing the low energy photons which only give you dose and the high energy photons which won't help with contrast.
- Rh/Rh is used for larger or denser breasts - because it's higher energy compared to Mo/Mo.
- Mo anode with Rhodium filter is also used to produce an intermediate energy spectra between a Mo/Mo and Rh/Rh combination.
- NEVER use a Rh Target (21 kev) with a Mo Filter (20 Kev K edge)
- Mo anode can also be combined with an aluminum filter, for a harder beam to penetrate denser breasts.
- Some digital systems will use Tungsten / Rho and Tungsten / Silver- this creates a higher energy spectrum for increased penetration and lower dose. Contrast is lost - but some post processing can bring it back.

Focal Spot Size: As mentioned before, mammo requires a better spatial resolution than you get with normal imaging. To achieve this you use a smaller focal spot:

High Yield:

- Mammography uses a focal spot of 0.3 and 0.1mm
- General X-ray uses focal spots of 0.6 and 1.2mm

Spatial Resolution

Screen Film Mammo 15 lp/mm
 Digital Mammo 7 lp/mm
 Digital Radiograph 3 lp/mm
 CT 0.7 lp/mm
 MRI 0.3 lp/mm

A smaller focal spot is not going to tolerate heat as well. To address this you have to use a lower mA (otherwise you'd melt the anode). The mA is limited to 50 for 0.1mm, and 100 for 0.3mm. Another issue is that since you are using a lower mA you need a longer exposure time. Generally you still try and keep the exposure time under 2 seconds.

Heel Effect: I mentioned this before. Just remember the cathode side goes near the chest wall. Also, the loss of energy on the anode side (nipple side) is compensated for by angling the tube up to about 20 degrees.

Effective Anode Angle — Anode Angle i Tube Angle

The Beryllium Window: Most diagnostic tubes use pyrex glass - this is NOT used in mammo because it causes excessive attenuation of the energies used in mammo. Instead a beryllium exit window is used.

Compression: The breasts are placed in compression. This does several good things:

- Reduced Thickness = Less Scatter = Lower kVp can be chosen
- Lower kVp and Less Scatter = Improved Contrast
- Reduced Thickness = Less mAs needed = Less Dose
- Breast Doesn't Move = Less Motion Artifact
- Breast Smashed Closer to Bucky = Less Geometric Magnification
- Less Motion and Less Geometric Mag = Improved Spatial Resolution
- Less Tissue overlap

Anti-Scatter Grid: Since the breast is placed in compression and you are using lower kVp (both of which intrinsically reduce scatter) a smaller grid ratio is used.

Mammo uses a 4-5 grid ratio (general x-ray uses a 6-16).

Dose with the Grid - The grid removes scattered photons - which reduces some dose. However, the same technique can't be used with a grid or you will underexpose. So, the technique is turned up. Ultimately the dose is INCREASED with a grid.

"The Bucky Factor" - The higher the grid ratio the more you have to turn up the juice. 2x dose = 2x Bucky Factor.

Bucky Factor is 2 for Mammo and 5 for general x-ray

Magnification: Magnification is done with a smaller focal spot, a smaller paddle, and no grid (air gap technique is used instead).

Contact Mode vs Magnification Mode	
Contact Mode - The normal Mammogram	Magnifications — 1.5x — 2x
Breast is in direct contact with the bucky	
The Grid is on	No Grid - <i>Air Gap used to reduce scatter</i>
Larger Focal Spot - 0.3mm	Small Focal Spot 0.1mm
Regular Paddle	Smaller Paddle
	Increased exposure time

Testable Trivia: If you increase the air gap, you will increase the magnification.

Testable Trivia: If you increase the air gap, you will also increase the dose (because of the automatic exposure rate control ramping up the setting to compensate for less photons hitting the detector).

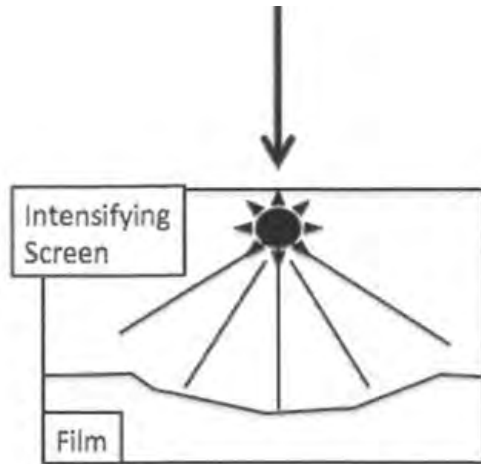
Film/Screen Combination:

Mammo uses a single emulsion film - matched to a single intensifying screen in the cassette. There are numerous benefits to this.

<i>Pros and Cons of the Single Emulsion</i>	
Pros	Cons
Less Parallax	Increased Dose
Less Crossover	
Better Spatial Resolution	

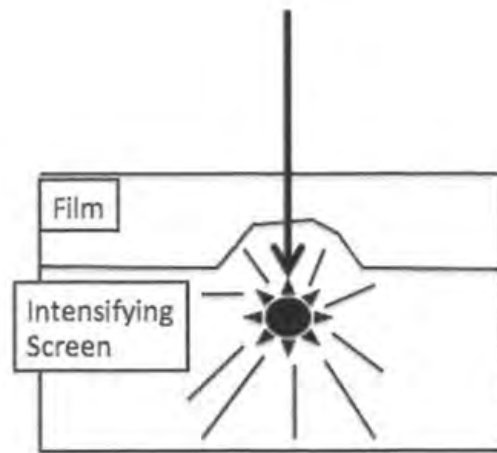
Orientation within the Cassette and Halation:

How the screen and film are oriented inside the cassette can affect spatial resolution. Bottom line is you want the film on top of the intensifying screen.



X-Ray -> Screen First
Light will spread out "Halation"

This increases the **Blur** (less spatial resolution)



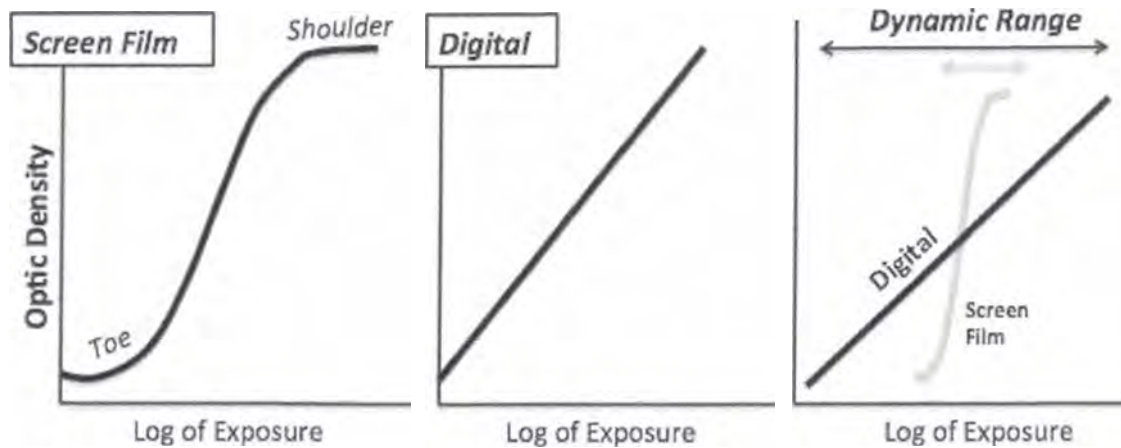
X-Ray -> Film First
Minimal Halation - Smaller Exposed Area
Improve Spatial Resolution

What is this "Screen" ? The "film screen" is a sheet of film with an emulsion on one side (or two - in mammo). The screen is basically a scintillator which takes x-rays and turns them into light. The actual x-ray film responds better to light from the screen, then a x-ray. The advantage of a screen is that you can use less dose (less exposure time). The down side is that lateral light diffusion within the screen will reduce your spatial resolution.

Testable Trivia: A screen does NOT reduce scatter.

What if you increase the thickness of the screen? A thicker screen will reduce your dose, but will also worsen the lateral light diffusion and therefore also worsen your spatial resolution.

What is this *optic density*? The actual x-ray film does NOT respond in a linear fashion to the light from the intensifying film. The classic look has a “toe” (caused by film base and fog), and then a shoulder. Digital systems has a pure linear curve.



Testable Trivia: Digital systems have a wider dynamic range with a linear characteristic curve (no shoulder and toe), compared to screen films.

Just Plain Trivia: Some Geezers refer to dynamic range as “latitude”

Processing Time: It’s much slower in Mammo. This extra time in the developer allows for more silver bromide crystals to develop and that can increase contrast and speed. Reduces patient dose.

Viewboxes:

- Mammo = 3000 cd/m²
- General Radiology = 1500 cd/m²
- Reading Room Light - Should not exceed 50 lux

Digital Mammography

Digital systems have the major advantage of allowing you to change the contrast and brightness after the acquisition of the image by adjusting the window and level.

Pixel Size and Spatial Resolution: Remember that smaller pixels = better spatial resolution. Additionally, you can lose spatial resolution from spread of electronic or light signal inside the digital or computed detectors.

- In general, most digital systems have lower spatial resolution (around 5-11 lp/mm) - relative to analog (11-13 lp/mm).
- Another point of trivia is that the **MQSA does NOT have specific line pair requirements for digital**; instead they are linked to the manufacturers specs.

Dose: Digital machines have 15% less dose compared to analog (1.8mGy for analog, 1.6 for digital). This is because the beam quality is better. Digital also has fewer repeat exams (because you can window and level). Also, the dose is not fixed - like it is with screens; you can turn the juice up and down as needed.

Noise Limited Images: The primary factor affecting the total noise is the number of photons interacting with the detector. Although digital images can have variable contrast, the noise is fixed after the exposure is taken.

Dark Noise: This is an additional type of noise from electronic fluctuations within the detector element. The effect is proportional to the temperature of the detector - that is why coolers are needed. You see these more with underexposed regions.

Flat Field Test: Imaging of a large piece of acrylic. This is done to improve image quality, and calibrate the digital detectors.

Digital Artifacts:

Ghosting: This is caused by a residual image from the prior exposure - burned into the detector. You see this when highly attenuating objects are placed in the beam. This is why *lead is not allowed on flat panel digital systems*.

Pixels Gone Bad: This can manifest as a square or a streak.

Comparing and Contrasting Mammo to General Dx - Summary

Mammo	General Radiology
Low Energy 25-35 kVp	High Energy: 50-120 kVp
Most Common Anode is Moly	Most Common Anode is Tungsten
Low Tube Current 100mA	High Tube Current 500mA
Long Exposure Times: 1000 ms	Fast Exposure Times: 50 ms
High Receptor Air Kerma 100pGy	Low Receptor Air Kerma 5 pGy
Beryllium Window	Pyrex Glass Window
Small Focal Spot	Larger Focal Spot
Lower Grid Ratio: 5-1	Higher Grid Ratio 10-1
High Optic Density	Low Optic Density
Brighter View boxes - 3000cd/m ²	Darker View boxes - 1500cd/m ²
Longer Processing Times	Shorter Processing Times

MQSA

Appropriate Target Range for Medical Audit	
Recall Rate	5-7%
Cancers/ 1000 Screened	3-8
PPV for Biopsy Recommendations	15-35%

Specific Tasks That You Should Memorize	
Processor QC	Daily
Darkroom Cleanliness	Daily
Viewbox Conditions	Weekly
Phantom Evaluation	Weekly
Repeat Analysis	Quarterly
Compression Test	Semi-Annually
Darkroom Fog	Semi-Annually
Screen-Film Contrast	Semi-Annually

Spatial Resolution and the Line Pair Phantom:

- *RSNA says: 13 LP/mm in the Anode - Cathode Direction*
- *RSNA says: 11 LP/mm in the left-right direction*
- *Hada Says: MQSA requires a resolution of 12 line pairs per mm for screen-film, and manufacturer specs for digital (~ 7 lp/mm).*

Mean Glandular Dose:

The MQSA has some breast phantom which is suppose to be an “average breast.” This thing is 4.2cm of compressed breast that is 50% adipose and 50% glandular.

The measured dose is with a grid.

Testable Trivia: Dose under 300 millirads (3mGy). This required dose is ONLY for the phantom, not a real human breast. There is no actual regulation for what a human breast can endure.

I myself dabbled in pacifism once

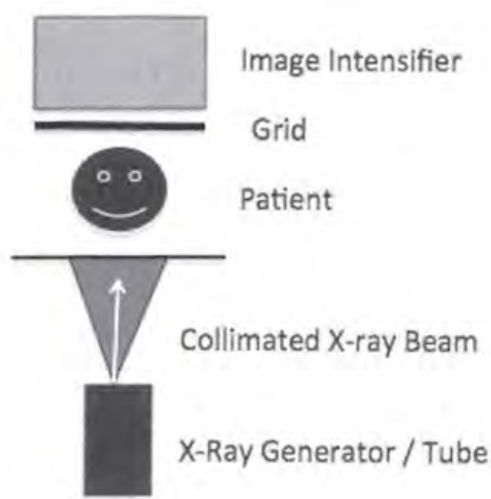
Section 5: Fluoro

Fluoro is different than regular diagnostic radiology. Radiographic images are static, and fluoroscopic imaging can be performed to view dynamic processes like swallowing or blood flow.

Regular Dx	Fluoro
mA 200-800	mA 0-5
kVp 50 -120	kVp 50-120
Very short exposure times	Longer exposure times
Focal Tube Spot 1.0 -1.2mm	Focal Spot 0.3-0.6

Things to Know:

- * *Regular Dx - done with 200-800 mA, with very short exposure times*
- * *Fluoro - much lower mA, with longer exposure times (this reduces the chance of overheating, so it can stay on longer)*
- * Small (0.3-0.6) Focal Spot is used for Fluoro - in order to limit geometrical blurring
- * Larger Focal Spot is used for the “Spot Image” - because greater tube current is needed
- * A “Spot Image” is the same thing as a conventional x-ray
- * A Fluoro Frame Shot has more quantum mottle than a Spot Image (less photons)
- * Fluoro uses a Grid - just like regular x-ray 10:1



What does the Collimator do?

Multiple sets of shutter blades define the shape of the x-ray beam. By “coning down” the beam, you get less scatter and better image contrast. It also reduces dose.

What does the Image Intensifier do?

Converts the x-rays to electrons, accelerates them, and then converts that to a visible image.

Fluoro systems can be used in GI and GU radiology.

- *GI*: Camera below table
- *GU*: camera above table - bladder closer to image receptors; reduces focal spot blur
 - They have higher operator lens dose

WTF is “Quantum Mottle” ?

It is the most important source of noise in radiography. It depends on the number (concentration) of x-ray photons used to produce an image (more photons = less mottle). It's apparently too complicated a concept to really understand much past this, other than to say it's caused by the statistical fluctuation (standard deviation) of the number of quanta of photons per unit size absorbed by the screen.

- Increasing the speed (kvp) increases the mottle
- Increasing the number of photons decreases the mottle

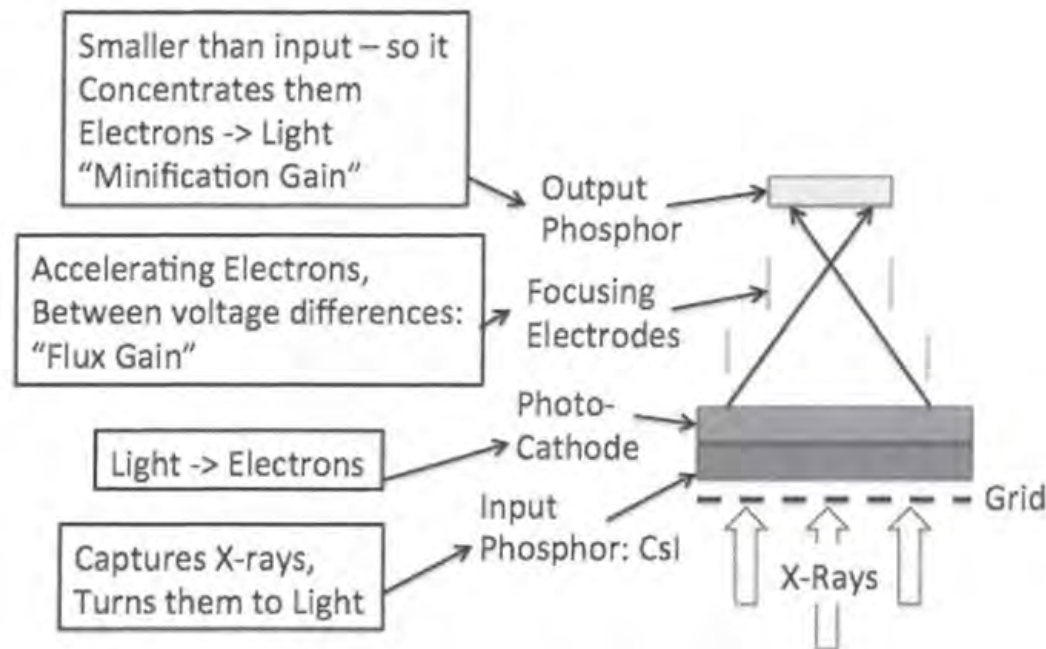
Image Receptor Types:

There are two kinds:

- (1) Image Intensifiers (I.I.) - the old kind
- (2) Flat Panel Detectors (F.P.D) - the new kind

Image Intensifier:

Image Intensifier



The purpose of an image intensifier is to convert x-rays to light and then back to electrons. Along the course of this conversion, the electron flux and energy are amplified by the image intensifier with the end result being a greater amount of light emerging from the output phosphor. To actually view the pictures, you must couple an image intensifier through lenses to a TV camera.

"Flux Gain" This describes the increase in magnitude of light coming from the output phosphor, relative to the input. This increase in magnitude is accomplished with a high voltage between the photocathode and the output phosphor (25-35 kV). The voltage causes electrons to accelerate and yields a gain in energy.

"Minification Gain" - Electrons from the large photocathode surface are concentrated on a *small output phosphor*. This increases the number of electrons per unit area, and therefore the energy per unit area.



Q: When you “Mag” something you are actually doing what?

A: Minifying it less

“*Brightness Gain*” - This is a term describing the increase in emitted light from the 1.1. compared to the amount of light you would have obtained if the 1.1. was never invented (and you just directly exposed the phosphorescent screen).

Testable Point: Brightness Gain is due to the combined effects of Flux Gain and Minification Gain.

$$\bullet BG = Flux\ Gain \times Minification\ Gain$$

“*Conversion Gain*” (Gx)- This is a term describing the efficiency of an 1.1. in changing incident x-rays into light at the output surface. *The older an 1.1. is, the more it sucks at this.*

Dealing with an Elderly I.I.

Solution 1: Use an aperture with a large hole in it. The down side to this is it increases the image noise.

Solution 2: Let the Automatic Brightness Control system do its job and crank up the juice (use more radiation). The down side to this is it increases the dose.

Solution 3: Put it in a nursing home, then get another one. The general rule for this is the Image Intensifier is usually replaced when the conversion gain falls to 50%. The down side to this is that it costs money to replace things, but it's ok you can offset the cost by cutting the Resident's benefits / salary....and hire a million \$/year VP or chairman to oversee the cost savings transition.

The Gains:

Flux Gain = Accelerating electrons between voltage differences.

Minification Gain = Electrons from large surface, concentrated on smaller surface

Brightness Gain = Light increase from the acceleration and concentration processes. Flux Gain x Minification Gain.

Conversion Gain: How good the 1.1. is at turning the electrons back into light.

Field-of-View: Collimation is electronically controlled with positioning changes made to irradiate a small region of anatomy - *you change to a smaller input surface*. The trick is that you are actually minifying the image less, as a smaller input surface is projected on the same output surface.

With a smaller input and unchanged output, the minification gain is decreased. If you did not change the dose, this decrease in minification gain would translate to less light from the output phosphor.

Geometric vs Electronic Magnification

Geometric Magnification - I touched on this earlier, but I want to make a few more points. To magnify something, you generally bring it closer to the x-ray source ($Mag = SID / SOD$). People usually think about the “inverse square law” for reducing radiation, but obviously it works the other way too. If you get closer to the tube you get more radiation— and it doesn’t double, it squares.

Electronic Magnification (Zoom) -

If you decrease the input field of view by half, then only one fourth of the input phosphor will be irradiated. If all other parameters are held constant, the brightness will also drop to one quarter. This gets the attention of the automatic exposure control which will ramp up the juice.

Testable Trivia: Both Electronic and Geometric Magnification Increase Dose. Geometric Magnification increases it more.

What is this “Automatic Brightness Control” ? This is a feedback circuit in the image intensifier / x-ray generator system that’s sole purpose in life is to maintain brightness at the output phosphor. It does this in a variety of ways; adjusting the kVp (mA fixed), adjusting the mA (kV fixed), or both (kV and mA) to maintain the brightness at the output phosphor. The consequence to the patient is an increase in dose.

Dose Compensation For Geometric Mag:

- (1) You can try and collimate.
- (2) You might be able to get rid of the grid, if you kept the receptor stationary' and moved the patient closer to the source. This would introduce an air gap, and naturally reduce scatter. Be careful how they word this question. That air gap is not gonna happen if you brought the tube closer, you need to move the patient closer to the tube and keep the receptor stationary.

Radiation Dose:

What regulates radiation dose? -Automatic Brightness Control System (ABC). It watches the light from the output phosphor and adjusts accordingly (increases kVp, increases mA, makes the x-ray pulse width longer, less x-ray filtration, or some combination).

What is the general radiation change with each magnification? -Increases dose by 1.4-2.0 times -You get less light out, the ABC turns up the juice to maintain the picture

Where is the ideal place to stand? On the same side of the patient as the imaging intensifier
-It is preferable to position the C arm x-ray tube under the table

Best Position of the I.I. and X-ray Tube? X- ray tube far away, with the I.I. close.

Having the image intensifier as close to the patient as possible does 3 things:

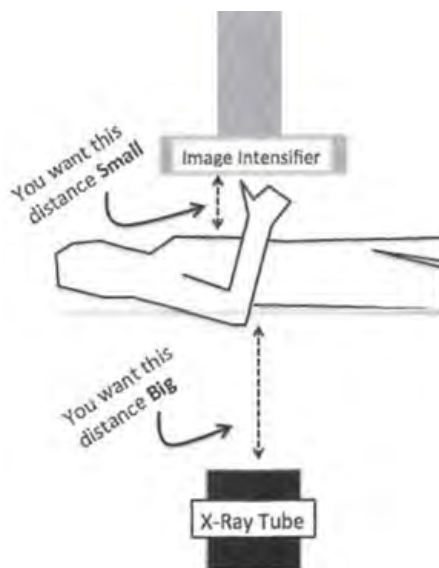
(1) Decreases patient dose.

- The ABC tries to make pretty pictures and more incident x-rays are detected when the I.I. is close so the ABC doesn't increase mA (to increase the number of x-rays generated) to make an adequate image.

(2) Decreases scatter to the operator.

- Less x-rays are made to scatter and less scatter escapes to irradiate the operator.

(3) Increases image sharpness. Decreases focal spot blur and magnification.



Raising the Imaging Receptor will ? Increases the dose - the machine will increase dose to compensate for the “source to image” receptor distance

Double the distance from the tube does what to dose?

- Decreases it by a factor of 4 (*inverse square law*).

A smaller field of view, does what to dose?

- With I.I. systems - increases it
- With FPD system - also increases it (usually), but doesn't have to

Does the order of mA and kVp adjust matter with regard to dose? - yes

- If mA is increased before kVp the dose gets higher
- If kVp is increased first the dose goes up less

WTF is the F# ?

F# =focal length/lens diameter

Lower F# = more light, faster lens, less pt radiation exposure

Effect of x-ray beam filtration on dose?

- More filtration - less low energy x-rays = less dose

How does adding an “Aperture” to an I.I system affect dose?

- An aperture with a smaller hole (larger F#) to block more light from the output phosphor results in a greater radiation dose rate to the patient compared with a system with a smaller F#.
- The idea is that, the size of the hole in the aperture should balance the amount of Quantum Mottle to an acceptable dose

How does kvp selection affect dose?

- Higher kVps result in more penetrating x-rays.
- Higher kVp's result in lower patient radiation doses

How good are lead aprons? When should you wear one?

- 1mm of Pb stops about 90% of radiation (it's not 100%)
- If you are within 6 feet of a fluoroscope you should lead up

Techniques to reduce dose to patient?

- Positioning away from the source
- Using the smallest field of view by collimating (this also improves resolution)
- Avoiding magnification

Seemingly Random Trivia

Various federal regulatory bodies limit the patient entrance dose rate to a maximum value of “87” **mGy per minute** (“10” R/minute) in the normal mode of operation.

A “*high level mode*” can be used, you just have to have audible or visual alarms (in addition to the normal time alarm used in normal fluoroscopy.)

In “*high level mode*”, the maximum patient entrance dose rate must be less than “174” mGy per minute (“20” R/minute).

“5-10” digital spot films = the dose of 1 minute of fluoroscopy (assuming same FOV).

Spatial Resolution

Q: What improves spatial resolution?

A: Magnification (less minification)

Q: What is the main limiter?

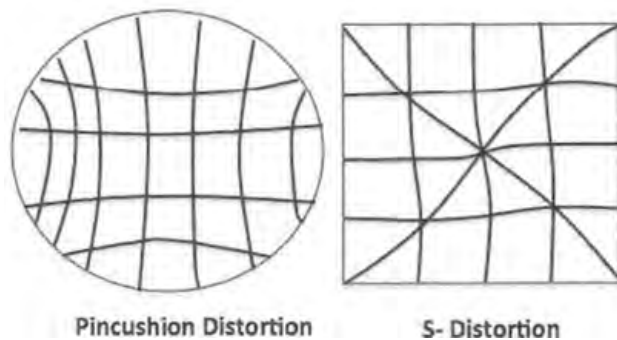
A: The quality of the display TV

Artifacts:

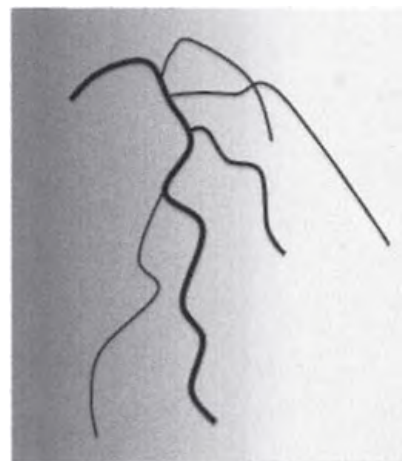
Pincushion Distortion: With a large field of view you can sometimes get the appearance of bent lines at the periphery (lines that should be straight). The inward bowing pattern is said to resemble a pincushion.

S Distortion: This is a similar artifact to the pincushion, also seen more in larger fields of view. The etiology with this artifact is an interference of the earth's magnetic field on the flow of electrons heading towards the 1.1.

Making it better? The addition of a mythical material called "mu metal" supposedly can deflect the magnetic field (and protect against vampires).



Flair or Glare Artifact: If you have a transition from heavy attenuation to minimal attenuation - you can see bright white "Glare" at the periphery near the decreased attenuation. This is from an overproduction of x-rays in this thin area, to compensate for the nearby thick area.



Flair / Glare Artifact

-Image becomes bright with transition to less attenuation

Lag Artifact: You move the I.I. and the ghosted image is still superimposed from the prior field.

Vignetting Artifact: Because distances from the focusing point to the outer phosphor tend to vary, with the closest path in the center and the farthest path at the edge you can end up with a dark periphery and a light center.



Vignetting Artifact
- *Edges are darker than center*

Saturation Artifact: If the dose is cranked up to try and penetrate a very dense object (classically metal) you can end up with regions around the metal appearing very bright.



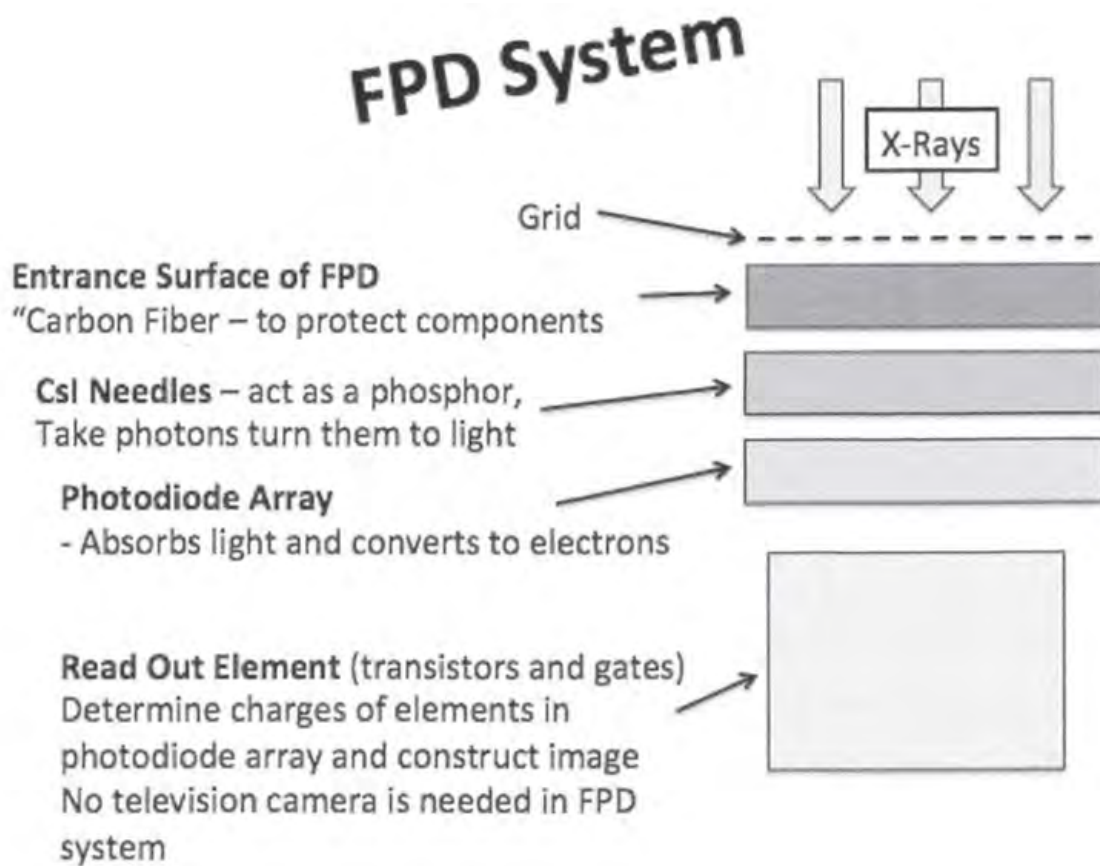
Saturation Artifact

Image Receptor Types:

There are two kinds:

- (1) *Image Intensifiers (I. I.) - the old kind*
- (2) *Flat Panel Detectors (F.P.D) - the new kind*

**We discussed the I.I. - Now let's talk about the F.P.D*

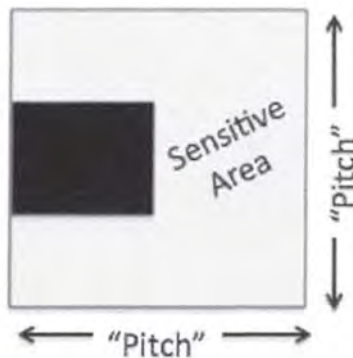


As you can see all that I.I. stuff doesn't happen with the modern system (oh, don't worry you will still get tested on it). There is some general vocab that I want to discuss, then the differences in artifacts between the two systems, then I'm going to end on spatial resolution - a high yield topic.

Vocab:

“Pitch” There is a thing called pitch. Essentially it is the linear dimension of a detector element. This is different than the “pitch” you think about with CT (more on that later).

“Fill Factor” There is a thing called fill factor. The system isn’t 100% efficient, only a portion is actual sensitive to light. The ratio of the sensitive area over total area is the fill factor.



$$\text{Fill Factor} = \frac{\text{Sensitive Area}}{\text{Pitch}^2}$$

As the detector element gets smaller you get better spatial resolution, but the “fill factor” decreases. In other words, a smaller detector element will have superior resolution but will require more radiation.

“Matrix” - The matrix is the number of detector elements (or pixels) on the surface of the FPD - in each dimension (horizontal and vertical).

1 Detector Element = 1 Pixel

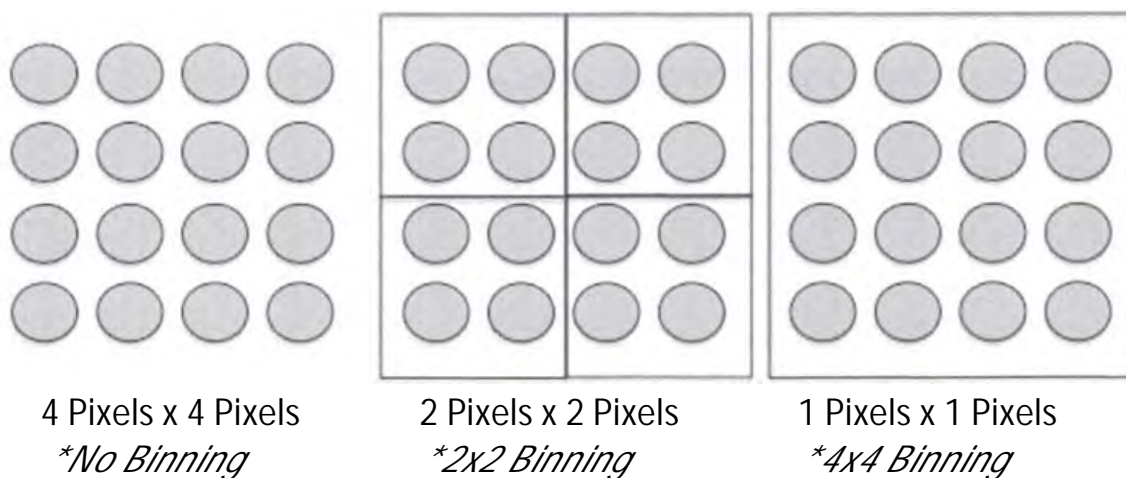
Math problem: If the matrix size is 1100 x 1100, and the FOV is 25cm, what is the pixel size (or detector element)?

the formula is: $\text{Pixel} = \text{FOV} / \text{Matrix}$.

But, I was trying to get sneaky with the units (a very common trick): 25cm = 250mm.

$$250 / 1100 = 0.23\text{mm}$$

Binning: There is a thing called binning. Taking several detector elements (DELs) and making a large DEL. The idea is the you reduce the amount of data (and reduce Quantum Mottle - less variation in x-ray photons from pixel to pixel).



- Less Mottle means you can reduce radiation and keep the same noise.
- Larger DEL does reduce the spatial resolution

When is “Binning” especially useful? -With large FOV (when there are too many pixels in the image)

If there is no “binning” the spatial resolution changes in what way from different fields of view (in a FPD system)? No change. If you have binning, large FOVs will have less spatial resolution (but can use less radiation to maintain the noise level). Remember “binning” is a FPD thing, not an 1.1. thing

What usually happens (without employing binning) with smaller FOVs? Dose is increased to reduce quantum mottle. If you employ binning, dose can be reduced. For example, If you combine 4 DEL you can reduce dose by 50%

“Frame Averaging (Recursive Filtration)” - This is an image process feature that adds several images together with different weight factors.

Pro: It’s done to reduce quantum mottle and increase the signal. SNR improves.

Cons: It increases the susceptibility to motion artifact and ghosting.

Artifacts (FPD):

Are artifacts seen with I.I.s the same as FPDs? Nope. **FPDs do NOT have pincushion, S distortions, vignette, glare, or saturation artifacts**

Bad Pixel - appear as either white or black spots. One method to correct for this (often built into the system) is to interpolate in order to fill the data.

Lag Artifact (Ghosting) can also occur with FPD. This tends to occur if the exposure uses very high radiation.

Spatial Resolution:

What limits of spatial resolution of an I.I.fluoro system? -The television display system. The resolution of a TV depends on raster (scan) lines, the bandwidth, and the FOV.

Does the display limit the spatial resolution of a FPD system? Nope, FPDs usually have displays with the same matrix as the image receptor

Vertical Resolution is limited by? The number of raster lines that the display monitor uses. You have to alternate white and black to reproduce a line pair image on the I.I. or FPD, so the max number of line pairs in the vertical direction is half the number of actual lines used. Because the bars in the line pair pattern may not totally line up with the raster lines, it's less than that so you need to correct with the Kell Factor (Kell Factor is some derived factor: 0.7).

$$\text{Vertical Resolution} = \frac{\text{Raster Lines} \times \text{Kell Factor}}{2 \times \text{FOV (mm)}}$$

So decreasing the FOV (mag mode) improves spatial resolution

How is horizontal resolution different than vertical resolution? The number of dots per line must be calculated indirectly from the bandwidth. The number of line pairs along the horizontal direction is half the dots in a raster line

If you measure spatial resolution at a diagonal (45 degrees to the raster lines) does it get better or worse? Better - spatial resolution improves by a factor of $\sqrt{2}$ over the vertical or horizontal resolution

Better pure spatial resolution FPD vs I.I ? I.I. systems are better, and change with FOV.

Spatial Resolution is Limited By:

FPD systems are limited by? - Detector element size (around 2.5-3.0 lp/mm)

I.I systems are limited by? TV systems (1.0-2.0 lp/mm for GI, and 2.0-4.0 lp/mm for angio)

8 Factors Affecting Spatial Resolution:

FOV - smaller FOV better resolution

Focal Spot Size - usually not an issue unless you get the anatomy away from the image receptor

Image Receptor Limitations - detector element for FPD and television for I.I

Motion and Temporal Factors - motion creates ghosting

Dynamic Range - This is only an issue for I.I systems - with variability in very dense or very transparent stuff

Pixel Binning - Binning increases pixel size - reduced spatial resolution (but improves SNR)

Frame Averaging - increases SNR (less mottle), more susceptible to blur

Pulsed Fluoro - Dose is administered in pulses, instead of continuously. You get less motion artifact (better spatial resolution in moving objects) and overall less dose.

QA

How is spatial resolution tested for? - Lead bar pattern

How is distortion checked for? - Use a mesh screen or plate. Look for straight lines (not pincushion or S distortion).

Fluoro in IR

Focal Spot: You are trying to visualize small things (tiny vessels in real time) - this means you need a small focal spot + large number of x-rays. To deal with this small focal spot, local heat exchanges must be added to avoid melting the whole unit. The anode angle is smaller when compared to conventional x-ray. The smaller angle leads to an increased heel effect. Fortunately, in IR the heel effect remains zero because you use a small field of view and small image detector (only allows the central portion of the x-ray beam to be imaged). Focal spots can be exchanged (large, small, micro) - depending on the need for resolution.

kVp: The best kVp to use with contrast is between 60-80 kVp (average beams hit that k-edge nicely). A higher kVp loses iodine contrast.

Filter: There is a thing called an “equalization filter” or a “soft filter.” These reduce intensity but do NOT completely block the beam. They are used to “taper” the radiation profile, and are often employed when imaging the leg, arm, or pediatric patients.

Grid: Since grids are usually used, the testable trivia is that they are often NOT used on extremities or peds.

Digital Subtraction Angiography (DSA) - This system works by taking a single frame mask image and subtracting it from another single frame contrast image - with the goal of removing anything that is not moving. This leaves the stuff that is moving (blood).

Patient Dose

- Most IR systems use a pulsed fluoro (helps reduce dose).
- 50% of the dose is delivered in the superficial 3-5 cm of skin/fat
- The depth of this 50% depends on the kVp and filtration (*higher kVp + Copper Filtration = more penetration*)
- For a body (or body part) measuring less than 10cm (a baby for example) - the grid should be off
- A thicker (fatter) patient gets more skin dose. This is because automatic brightness control sees less penetration - then cranks up the dose (higher kVp).
- Additional high dose situations: lateral and oblique views
- Patient and operator dose doubles with a lateral view (compared to PA)
- The typical dose is about 0.3 - 0.5 mGy per frame at the entrance skin position (1 Ox - 20x more per image than fluoro)
- Total Dose = (dose per frame) x (frame rate) x (duration x number of runs)

“Source to Skin Distance” (SSD) = how close the patient is relative to the x-ray source. If you are using under the table positioning the SSD depends on table height.

Small SSD = High Dose

Short Angiographers should stand on a platform (or be carried by a trainee) so that the source to image receptor distance is kept at 100cm or more.

“Dose Spreading” The idea here is to change the angle of the gantry (especially in a long case) in order to spread the skin dose over a broader area - decreasing the skin dose to any specific location.

“Best Place to Stand” You should try and stand / work on the image receptor side of the patient. You are trying to avoid the large amount of Compton scatter radiation produced where the beam enters the patient.

Dose Area Product (Kerma Area Product) This measures the radiation dose to air in mGy - multiplied by the collimator area - then reported in mGy/cm. **The measurement is independent of beam location.** It's true that as you move the beam away from the patient the intensity decreases- BUT, the beam spreads out more. These two things occur in equal amounts so the DAP (KAP) is NOT dependent on location.

DAP (KAP): Low Dose to Large Skin Area = High Dose to Small Skin Area
Magnification - will increase Air Kerma, but NOT KAP

Obviously this doesn't help you grade risk of a skin burn. Instead it is used to estimate total energy deposited in patient, effective dose, and cancer risk. If something reduces the DAP (KAP) it probably also reduces the scatter and the patient's dose.

Interventional Reference Point (IRP)- This describes the use of an ionization chamber with a set reference point (*15cm closer to the source than the isocenter of the IR system*) to measure radiation emitted from the source. Skin dose can be above or below this point.

Because IRP ignores geometry, table attenuation, and back scatter, it probably over or underestimates patient's skin dose every time - but it is currently the best thing available.

Testable Trivia: The dose (outside lead) standing 1 meter from the patient is about 1/1000 of the dose received by the patient.

Regulatory Doses:

- No High Level Control (HLC) Present = 10 R / min (87mGy/min)
- HLC on = 20 R/ min (176 mGy/min)
- During Image recording = no limit if pulsed

Skin Doses:

- Below 2 Gy - No action needed
- 2-5 Gy - advise patient to watch for burns - especially 10 days post procedure
- Above 5 Gy - procedure and dose should be reviewed by physics
- 2 Gy - Early Transient Erythema
- 3 Gy - Temporary Epilation (hair loss)
- 6 Gy - Chronic Erythema
- 7 Gy - Permanent Epilation (hair loss)
- 10 Gy - Telangiectasia
- 13 Gy - Dry Desquamation
- 18 Gy - Moist Desquamation / Ulceration
- 24 Gy - Secondary ulceration

Operator Doses:

- You get about 0.1% of what the patient gets - at 1 meter
- In 1 year - you typically get about 5 mSv
- Regulatory dose limit is 50mSv per year
- Conceptus dose limit is 0.5mSv per month
- Eye dose limit is 20 mSv/year (*this was recently changed from 150mSv/year*)
- Extremity dose limit is 500 mSv/year
- Lead glasses are helpful - plastic glasses are worthless

High Yield Points:

- * The tube goes under the patient, as far away as possible (largest SSD possible)
- * The receptor goes as close to the patient as possible
- * Use pulsed fluoro and collimate when possible
- * Move the beam around as much as possible (don't just bum up the dudes skin in the same spot)
- * Avoid electronic magnification as much as possible
- * No grid on babies or extremities

High Level Control

The maximum entrance exposure is 10 R/min in a conventional setting.

An option for high dose is "specially activated fluoro" or "high dose control", where you can have rates up to 20R/min.

The trick is an audible alarm must be on when this is used.

Calmer than you are dude

Section 6: CT

How does the CT scanner work:

Most CT scanners today are “3rd generation” - which means the x-ray tube and the detectors spin around the patient in synchrony. CT X-ray tubes use tungsten alloy targets placed on high speed rotating anodes. CT tubes are designed to operate at reasonable voltages (between 80-140 kV) with very high tube currents *up to 1000mA*. The typical focal spot is “large” measuring 0.6mm to 1,2mm. You need a large focal spot to handle all that power - 100kW.

The x-rays are filtered to remove low energy x-rays that would only increase dose.

Filtration Mechanisms:

- Copper or Aluminum (6mm) is used to filter the x-ray beam
 - Heavily filtered beam can have a half value thickness of up to 10 mm Al
 - Bow Tie filters are used compensate for uneven attenuation of the beam by the patient. These filters attenuate less in the center and more on the edges. Bow Tie Filters are made of low Z materials - like Teflon (to reduce hardening differences).
 - The x-ray tube anode-cathode axis is positioned perpendicular to the imaging plane to reduce the heel effect.
- Bow Tie Filters:*
- Compensate for uneven filtration
 - Reduce Scatter
 - Reduce Dose

Scatter Reduction:

- A collimator is used both at the x-ray tube, as well as the detector, with the purpose of shaping the x-ray beam (“*Defines the section thickness on a single slice*”). The collimator also reduces some scatter.
- Additional scatter reduction is further accomplished with “anti-scatter septa.” Which are grids, but you can’t call them grids - they are “septa!”

The CT fires off x-rays as it spins. The table moves the patient through. The detectors (highly efficient scintillation detectors) have a lot of information - which looks like a wavy mess (Sinogram). Then a bunch of math takes place (filter back projection), and you get a picture.

Detector Types:

There are two types of detectors; Scintillation Detectors (the modern type), and Gas Filled Detectors (the ones used back when dinosaurs roamed the earth). The Scintillator detectors are way more efficient and easier to produce.

3rd Generation Multi-slice vs Single Slice:

The number of detectors in the axial direction determines the number of slices that can be simultaneously acquired. The MDCT can acquire images with “isotropic resolution,” - which means they can do non-axial reconstructions without stretching pixels.

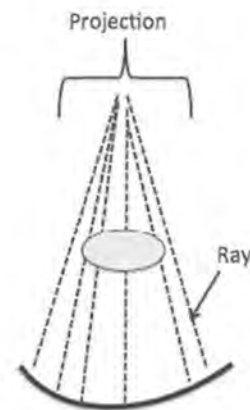
High Yield Point: Minimal slice thickness is determined by detector element aperture width in a modern CT.

Vocabulary:

What is this “Ray” - A “ray” is a measure of total x-ray attenuation along a line from the focal point to a single detector

What is this “Projection” - A “projection” is all rays at a given angle of the x-ray tube. In other words, it’s a “series of rays that pass through the patient - at the same orientation.

What is this “Sinogram” - A “sinogram” is a bunch of squiggly lines that represent the data from all the projections of all the tube angles (0-360).



What kind of x-rays are used with CT? Highly filtered, High kV (average energy 75 keV)

How is the image actually produced? Generating an image from the acquired data involves figuring out the linear attenuation coefficients of each pixel in the image matrix.

Back Projection - This was the original way. Equal attenuation was given to all pixels along a ray.

Filter Back Projection - This is the more modern way. By multiplying projections with a “mathematical filter,” you make a better picture.

Adaptive Statistical Iterative Reconstruction (ASIR) - “Forwarded” information is compared to actual information and differences are used to correct the image. What you need to know is (a) it can correct for noise, so you can (b) use a lower dose. It requires some major computer power, that’s why older systems couldn’t use it.

What is the matrix size for CT? Each Pixel is ? The matrix is 512 x 512, with each pixel representing 4096 possible shades of gray (12 bits).

Wait? How did you get that? $2^{12} = 4096$

What is the relationship between pixel width and height to voxels? They are the same. $\text{Pixel } W \times H = \text{Voxel } W \times H$. The difference is a voxel has a 3rd dimension (depth), which represents the slice thickness. A voxel is a cube, a pixel is a square.

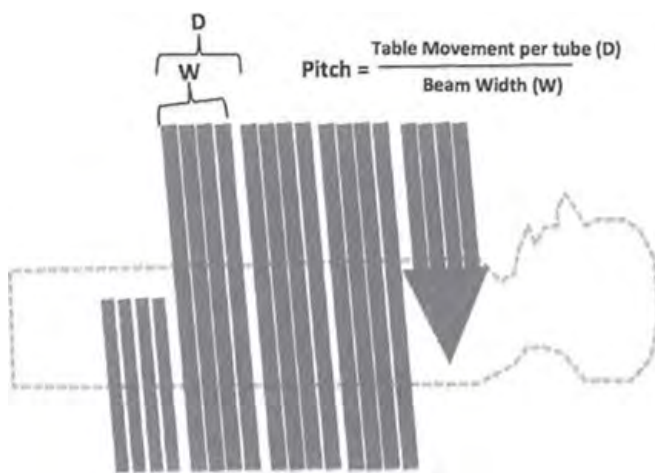
How do you calculate pixel size ? You divide the Field of View / Matrix Size

How do you improve spatial resolution ? You need to make the pixels smaller (matrix larger). Remember that $\text{Pixel Size} = \text{FOV} / \text{Matrix}$

Is mAs the same on CT as it is on plain film? The conventional definition of radiographic mAs is not useful in spiral CT. You have to use “effective mAs”. $\text{Tube Current (mA)} \times \text{Length of time that a given point in the patient is in the beam}$. Basically, the exposure time Obviously the exposure time is going to be related to the collimated beam width and table speed.

If you turn down the mAs, what happens to your images? Less mAs = More Noise

What is this “Pitch” ?



Understanding Pitch:

In this example, the pitch is greater than 1 because there is a gap between slices.

If the pitch is 1 the beam = the distance the table turns in one revolution (no gap).

If the pitch < 1 than you have overlap (which increases dose).

Practice Math Problem:

- Calculate Pitch: Table Moves 12mm, Beam Width is 8mm; $12 / 8 = 1.5$
- **All things being equal - increasing the pitch does what to the radiation dose?** Reduces it (less overlap)

The Hounsfield Unit: The attenuation of other tissues is a “relative attenuation” - based on a comparison to water. Water is always “0”. The formula used to calculate HU is:

$$HU = 1000 \times (\text{attenuation of material} - \text{attenuation of water}) / \text{attenuation of water}.$$

What is the relationship between HU and X-ray attenuation? When HU increases by 10 HU, x ray attenuation increases by 1%

Increasing the Beam Width Through the Collimator:

- Reduces Scan Time (larger coverage with 1 turn)
- Reduces Motion Artifact (less scan time)
- Increases Partial Volume (more divergent beam)
- Does NOT change the radiation dose (mAs is unchanged, even though the scan time is less, a larger area of tissue is scanned at the same time).

Axial vs Helical Modes

Axial

The table is stationary. The tube comes on and takes a picture. The tube shuts off and the table moves up a slice. The tube comes back on and takes another picture

Advantages:

- Better spatial resolution on the z-dimension since full images sets are taken. No partial volume effect along the long axis. •
- The artifacts of partial volume with helical CT are noticed more along a curved surface. *Classic example = skull.*

Helical

The table moves at constant speed. The tube is on the entire time.

Advantages:

- Primary Advantage = Way faster
- Secondary Advantage = Post acquisition flexibility in the selection of slice location and lower probability of anatomic discontinuities between adjacent slices containing moving anatomy in the chest/abdomen scans

Fixed and Variable mA

Old scanners had fixed kvp and mA

New scanners can adjust via two methods

- (1) Scout image - to estimate density
- (2) on the fly with continuous modulation

Advantages are reduced radiation and more uniform signal to noise

Spatial Resolution

Spatial Resolution - This is the ability to distinguish small objects that are close together (tell that they are separate). Practically a bar pattern is used to measure this (line pairs per cm).

Spatial Frequency - number of line pairs per cm. This is inversely to the object size. Small objects have high spatial frequency, large objects have low spatial frequency.

Factors that affect spatial resolution:

Focal Spot Size

- A larger focal spot, means the object details are spread out over several detectors. This degrades and blurs the image
- Smaller focal spot = better spatial resolution

Magnification

- More magnification blurs the image

Detector Aperture Size

- As the detector size is reduced the cranial-caudal resolution increases
- The in plane "x-y axis" is NOT affected by aperture size.

Number of Projections

- More projections, more data. Better Resolution

Reconstruction Slice Thickness

- The thinner the detector element aperture, the better the spatial resolution in the Z direction

Spatial Resolution as a function of pixel size and display field of view:

- “Display Field of View” (DFOV) = Space defined by the user based on the anatomy size to be displayed. It is always less than (or equal to) scan field of view.
- Remember that Pixel Size = DFOV / Matrix Size. So increasing the matrix size or decreasing the DFOV will make your pixel size smaller. Smaller pixels = Better Spatial Resolution.

Spatial Resolution as a function of Pitch:

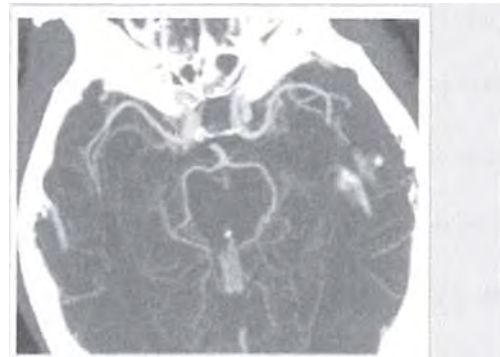
- As pitch increases, so does the width of the slice sensitivity profile (SSP).
- As the SSP widens the slice thickness increases. Thicker slices = Less Spatial Resolution.

Patient Motion

- Voluntary and involuntary movement leads to blurring. Blurring = reduced image resolution

Which **Vessel** is on Top?

At the institution I trained at, a popular quiz was to show a first year resident a MIP CTA of the brain and then say “which is on top, the basal vein of Rosenthal or the PCA?”



The question was a trick to illustrate that MIPs (maximum intensity projections) don't give you depth, they give you attenuation information. A vessel with a higher attenuation will appear continuous crossing a lower attenuation vessel (making it appear more superficial).

Contrast Resolution

Contrast resolution can be defined as the ability to discriminate small differences in object density from its surroundings for a specific target size and radiation dose (noise).

Dose: As the number of x-ray photons increases, the signal detected by the scanner increases, improving contrast resolution. However, radiation dose increases in proportion with photon flux.

Quantum Noise: Directly dependent on the number of x-ray photons. As the number of x-ray photons doubles, then, the signal increases by a factor of two, while the noise increases by a factor of $\sqrt{2}$.

- X Ray Double = Signal Double
- X Ray Double = Noise Increases by factor of $\sqrt{2}$

Signal-to-Noise: Contrast resolution improves with SNR. If the number of x-ray photons is doubled the signal doubles. If the x-rays double the noise increases by a factor of $\sqrt{2}$. So you have an increase of $2/\sqrt{2}$. In other words, as the signal (and proportionately the dose) increases, contrast resolution increases, but at a lesser rate.

Slice Thickness: Small slices = less photons per slice = less contrast resolution (the image is noisier). However, smaller slices = less partial volume averaging = improved spatial resolution.

Pixel Size (*Spatial Resolution vs Contrast Resolution*):

- Holding matrix size constant and decreasing FOV will decrease pixel size. This increases spatial resolution but decreases contrast resolution (less photons per box)
- Holding matrix size constant and increasing FOV will increase pixel size. This decreases spatial resolution but increases contrast resolution (more photons per box).

Factors That Affect Spatial Resolution	Factors That Affect Contrast Resolution
Focal Spot (smaller spot = better)	Number of X-Rays (mAs, kV, pitch). More dose (less mottle) will improve contrast resolution.
Detector Width (smaller detector = better)	Slice Thickness (thicker = more x-ray quanta = less noise).
Nyquist Limitations "Sampling" (Oversampling = better)	Reconstruction Method (Iterative > Filtered Back)
Reconstruction Filter (example - bone algorithm gives a higher spatial resolution)	Reconstruction Filter (Soft tissue > Bone)

Key Points

- Most CTs have a kVp of 120
- Peds and Skinny people have kVp of around 80 (*reduces dose and increases image contrast*).
- Fat people need larger kVp.

Cardiac Imaging

Cardiac imaging is best performed during diastole. There are two main methods; prospective and retrospective - I'm certain the differences in the two would make good multiple choice questions.

-Prospective: "Step and Shoot" - R-R interval

- Pro: There is reduced radiation b/c the scanner isn't on the whole time
- Con: No functional imaging
- Trivia: Always axial, not helical

-Retrospective: Scans the whole time, then back calculates

- Pro: Can do functional imaging
- Con: Higher radiation (use of low pitch - increases dose)
- Trivia: this is helical

CT Fluoro

- Near real time imaging, with the CT image constantly updated (6 per second).
- Low tube currents (20-50 mA) are used to minimize radiation doses.

Dual Energy

At a single photon energy is it possible to tell two different materials apart? Nope

How does dual energy CT Work? -Scan is acquired using both 140 and 80kvp (instead of just 120). The H.U. of each pixel is obtained at both energies. The image is dirtier, but you can do all kinds of stuff like characterize the material (*what is the renal stone made of?*), or do a virtual non con. It all has to do with different atomic numbers absorbing photons differently.

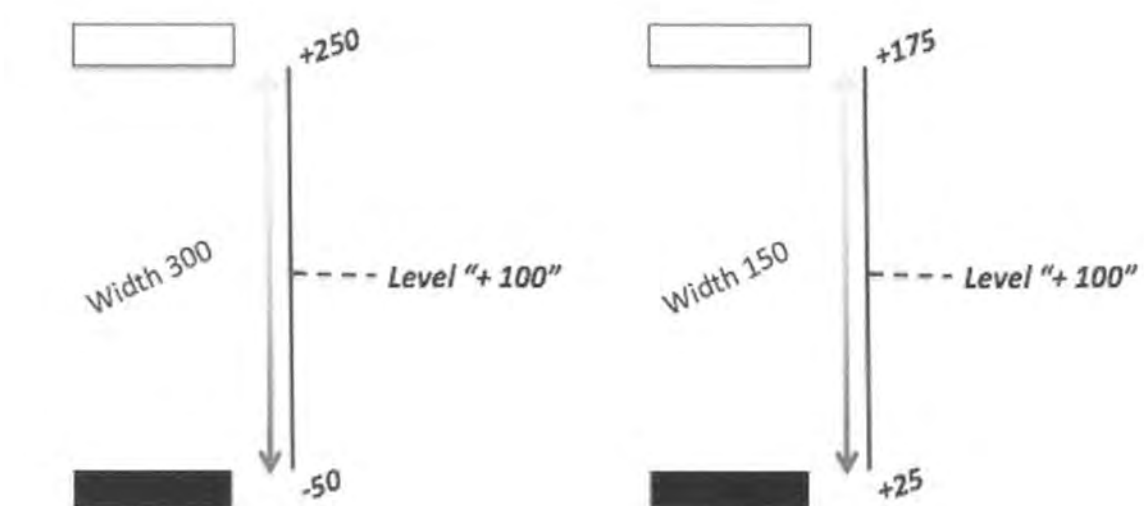
CT Window Width and Level:

It's a basic concept that is highly testable. The "Level" is the midpoint of the gray scale display (the "center"). You want your level at the attenuation of the thing you are interested in. For example, if you are interested in bone - you want a high level. The width is selected based on what you are comparing. If you are comparing things with very different densities you want a wide width. If you are comparing things with very similar densities (example white and gray matter), you want a very narrow window width.

How this concept can be tested? Really there are two main ways: (1) you can ask a typical window for lung, bone, liver etc... This is partly memorization (see chart) but you can sorta figure it out based on the principals I mentioned above. (2) They can ask you below or above what level will give you a white or black reading.

This kind of question is easiest to solve if you draw it out. For example, if your level is set at 100 and you width is 300, you will have 150 above and 150 below at some gray scale (150 is half of 300). In this case, above 250 will be seen as solid white and below a -50 will be seen as black. A second example shows the same thing using a more narrow window of 150.

Window , Level in HU	
Brain	W 80, L +40
Lung	W 1500, L-400
Abdomen	W 400, L+50
Bone	W 1600, L+500



Radiation Dose Measures: CT Specific

“Variation within the Scan Plane” - In regular Dx imaging, the entrance skin dose is much larger than the exit skin dose. In CT, because the scanner spins 360 degrees - the dose is symmetrically distributed. Obviously, the center is still gonna get less (body phantoms show it to be about 50% less in the middle - compared to the edge). When you compare a head to a body phantom the center dose is similar to the skin (smaller diameter).

Head Scans: Central and Surface are very similar

Body Scans: Surface is about twice the central dose.



“ZAxis Variation ” - There are often “tails” of radiation along the edge of the area being scanned. As a result, the profile of radiation is not limited to the primary area being imaged. If multiple scans are performed these tails add up with the original scan.

“CTDI”- CT Dose Index - This is the radiation dose, normalized to beam width. There are subtypes - the difference of which could easily be tested on a multiple choice test.

- “Weighted CTDI” - This is $\frac{1}{3}$ the central CTDI + $\frac{2}{3}$ the Peripheral CTDI (expressed in mGy)
- “Volume CTDI” - This is obtained by dividing weighted CTDI by the Pitch. Remember that Weighted CTDI is the intensity being used and can relate to mottle.

Quick Review of Pitch

- Doses in helical scanning with a pitch of 1.0 are similar to those from axial scanning.
- If the pitch is < 1.0 then the dose increases because the slices overlap.
- If the pitch is > 1.0 then the dose decreases because energy is more spread out.
- The relationship is proportional. A pitch of 2 halves the dose. A pitch of 0.5 doubles the dose.

“DLP” - Dose Length Product - This value is simply the CTDI - Vol x the length of the scan in cm.

$$\text{DLP} = \text{CTDI}_{\text{vol}} \times \text{Scan Length}$$

“Effective Dose” for CT: Effective Dose = k x DLP. Remember that “k” is a body part constant. Effective dose is going to be in Sv.

Phantom Size - These CTDI numbers are based on phantoms. The body phantom is 32 cm in diameter. If the patient is larger than the phantom, then dose is over estimated. If the patient is smaller than the phantom, then dose is under estimated.

Average Dose (CTDI - in mGy) Corrected effective dose is different

ACR Established Diagnostic CT Reference Values.

- Adult Head = 58 (effective dose 1-2 mSv)
- Adult Abd = 18 (effective dose 8-11 mSv)
- Peds Abdomen = 15

CTDI_{vol}:

- 75 mGy for Head,
- 25 mGy for Adult Abd,
- 20 mGy for Peds Abd (5 year old)

There is a “Reference dose” set by the ACR at 75 percentile - doses above that should be “investigated” and reduced if possible.

Risk of radiation induced cancer per dose?

- 5% per Sv = Adult
- Up to 15% per Sv for Child
- About 1/1 Oth that for someone older than 50

Pediatric Considerations:

- It’s recommended that you reduce mAs.
- Reduced techniques are possible because x-ray penetration is greater in children
- Dose Reduction in head CT are more modest than peds belly

Strategies to reduce dose to the breast:

- Do the scan at reduced mA (problem is the images look like shit)
- Use a milliamperage modulation (adjust based on density) * this is the preferred method
- Shield the breasts with bismuth - you get artifact and a degraded image (beam hardening can falsely elevate H.U. directly deep to the shield.

Dose Related Trivia:

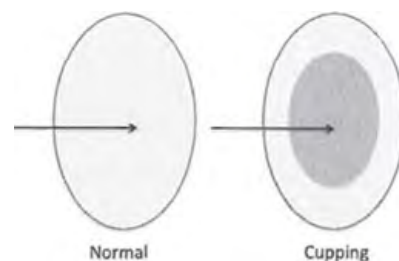
- Dose of 1 Chest CT is about equal to 100 PA + Lateral CXRs
- CT of the extremities has a very low effective dose (< 1 mSv) because they don't contain any radiosensitive organs.
- Embryo dose in CT A&P is around 30 mGy.

Individual dose monitoring is mandated if the occupational dose is favored to be greater than 10% the annual dose limit (500 mrem).

CT Artifacts:

Beam Hardening- As the x-ray beam passes through an object the lower energy photons are removed preferentially, leaving a "harder beam" with an increased average energy. There are two artifacts associated with this.

Cupping - The x-rays passing through the middle of a uniform shape (like a head) are hardened more than those traveling through the periphery (a shorter path). The harder the beam the slower the rate of attenuation. The manifestation is the center of the image appears darker than the peripheral portions.



Dark Bands /Streak - This occurs in the setting of two dense objects. X-rays that pass through one are less attenuated than those that pass through both. The result is dark bands and streaks between those two objects. The classic location is bone or where a dense contrast was used.

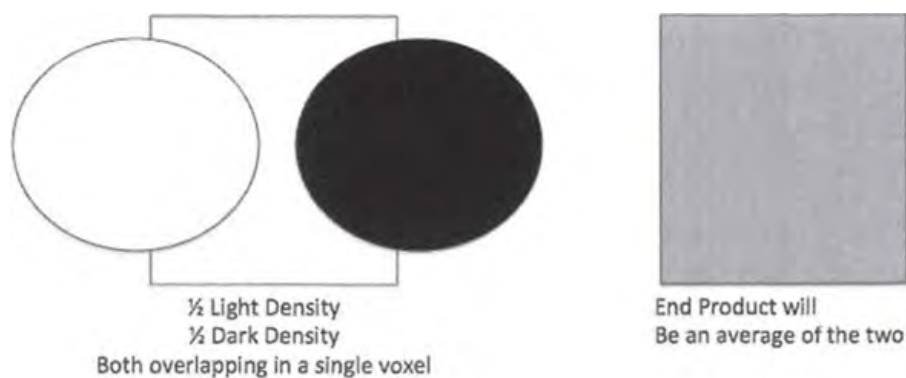
Fixing Beam Hardening:

- (1) Filtration - Pre hardening of the beam, to remove lower energy components before it hits the patient, and/or the addition of a bow-tie filter.
- (2) Calibration Correction - Using a phantom to allow the detector to compensate for hardening effects.
- (3) Correct Software - An iterative correction algorithm can be used.
- (4) Avoidance - Tilt the gantry or position the patient to avoid areas the cause hardening.

Partial Volume: This can occur in two main ways.

Pattern 1 ("Partial Volume Effect"): A dense object protrudes partially into the width of an x-ray beam. This results in divergence of the beam and manifests as shading artifacts adjacent to said object.

Pattern 2: CT voxels are 3D cubes. If you have a dense thing taking up half the cube, and a sparse (low attenuating) thing in the other half of the cube the machine will average the two together giving you something that has intermediate density. The classic location is the skull base averaging with CSF or brain to sorta look like blood.



Fixing partial volume: Make your slices thinner. If the noise is a problem, acquire thin slices then generate thicker slices by adding them together.

Photon Starvation: High attenuating areas (classically the shoulders) can result in photon starvation manifesting as streaking. It's seen when the beam travels horizontally - through the greatest area of attenuation.

Fixing Photon Starvation: There are two main ways to fix this. (1) Automatic tube current modulation. If you increase the dose through the area of greater attenuation you can add enough photons to overcome this effect. (2) Adaptive filtration can be performed to correct the attenuation profile "smooth the data" in the high attenuation portions.

Under Sampling: An insufficiency number of projections used to reconstruct the CT can diminish quality, and result in mis-registration artifacts. There are two main types.

View Aliasing: This is when you have under sampling between projections. You see fine stripes radiating from the edge (but at a distance from) a dense object. This is fixed by acquiring the largest possible number of projects per rotation - slowing the rotation speed.

Ray Aliasing: This is when you have under sampling within a projection. You see strips appearing close to the structure. This is fixed by using specialized high resolution techniques - manufacturer employed.

Metal Artifact: Metal causes streak artifact. It does this through several mechanisms: beam hardening, partial volume, aliasing, and having density ranges higher than what can be handled by the computer. Testable trivia is that metals with high Z (Iron, Platinum) tend to have more artifacts than those with lower Z (Titanium).

Fixing Metal Artifact: Tell the patient to remove it. Increase the kVp (sometimes works). Use thinner slices. Certain interpolation software can help.

Patient Motion: Motion can cause misregistration - which manifests as shading or streaking, especially on reconstructions.

Fixing Motion Artifact: Tie the crazy patients down. Use a modern (fast) scanner. Align the scanner in the primary direction of motion (vertically above or below a chest scan - for breathing). "Over scanning" an extra 10% on the 360 rotation, with the repeated portion averaged. Gating - like in cardiac.

Over-Scanning vs Over-Ranging

Over-Scanning - Essentially having a pitch < 1.

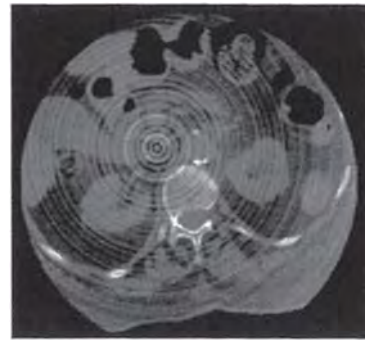
Over-Ranging - Scanning above and below the target, to collect additional data for a helical scan.

Incomplete Projection - If parts of the patient are hanging outside the field, but still attenuating x-rays this messes with the computer's math. Examples include, arms hanging down or having the IV contrast on or near the patient.

Fixing incomplete projections: Position the patient correctly.

Ring Artifact: A calibration error or defective detector on a third generation scanner will cause errors in angular position - resulting in a circular artifact.

Fixing Ring Artifact - Recalibrate your dinosaur detector, or replace the broken part.



Ring Artifact

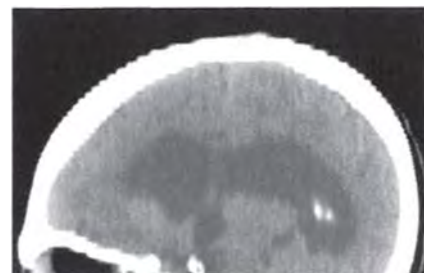
Helical Artifact in the Axial Plane: Single Section

You get the same artifacts with helical scanning modes plus you get some additional artifact from helical interpolation. The main place you see this is around the top of the skull (anatomy changing rapidly in the Z direction). The higher the pitch the worse this is. To minimize these artifacts you have to reduce the variation in the Z direction - use a low pitch, use 180 degree instead of 360 when possible, and using thin sections instead of thick. Testable trivia - this is why head CTs are still commonly done with axial scanning over helical.

Helical Artifact in Multi-Section

Distortion is more complicated on multi-section scanners, with a classic “windmill” appearance where several rows of detectors intersect. It worsens with increased helical pitch. A “Z-Filter” is done to reduce the severity of windmill artifacts.

Stair Step Artifact: This is seen as a “stair step” on the edges of a multi-planar reformatted image, when you have a wide collimation of non-overlapping intervals. It’s less severe with a helical scanner - where you are getting some overlap. It’s fixed by making thin slices.



Stair Step Artifact

Zebra Artifact: This is another reformat artifact, which can occur from helical data - secondary to the helical interpolation process (increases noise along the Z axis). The effect manifests as stripes (like a zebra) most pronounced on a 3D image. The effect is most significant away from the axis of rotation - noise is worst off axis.

It's a league game, Smokey

Section 7: Ultrasound

Sound is not light. Sound requires a medium to travel in. It's best to think of sound as a mechanical energy that produces vibrations when propagating through material. These vibrations produce alternating areas of:

- High Pressure (Compression) and
- Low Pressure (Rarefaction)

Frequency: This is the rate of change between compression and rarefaction - given in Hertz. In other words, its the number of times the wave oscillates through a cycle each second.

Wavelength: The distance between areas of compression.

Remember this from general chemistry? $Speed = Wavelength \times Frequency$

Speed is thought of as being constant (in a particular medium), so that an increase in frequency decreases the wavelength - and vice versa.

Speed in Different Materials: This is based on the compressibility of something. Things that are very compressible (air) will have a very low speed. Things that are not very compressible (bone) will have a very fast speed. The ultrasound machine is stupid - and just assumes everything travels at 1540 m/s in tissue. The fact that the machine thinks speed is constant is a source of artifacts (discussed later).

Speeds effect on frequency - None. The frequency is the same, irrelevant of the sound speed in various media. Therefore the wavelength changes in media.

Wave Interference Patterns: Modern ultrasound machines make a bunch of different sound beams. These sound beams can either have “constructive effects” and help each other - increase the amplitude, “destructive effects” and hurt each other - decrease the amplitude, or complex effects and be complex. These interactions are important in shaping and steering the beam.

Relative Intensity - The dB. A change of 10 in the dB scale corresponds to two orders of magnitude (100 times) and so forth. The dB is based on a log 10 scale.

- Reducing the sound intensity to 10% is -10 dB
- Reducing to 1% is - 20 dB
- Reducing to 0.1 % is - 30 dB

Testable Point: A loss of 3 dB (-3 dB) represents a 50% loss of signal intensity (power).

Testable Point: The tissue thickness that reduces the ultrasound intensity by 3 dB is considered the "half-value" thickness.

Interactions of Ultrasound with Matter

Ultrasound interacts with matter by:

- Reflection
- Refraction
- Scattering
- Absorption

Reflection: Ultrasound energy gets reflected at a boundary between two tissues because of the differences in the acoustic impedances of the two tissues. A large difference in “stiffness” results in a large reflection of energy.

Impedance: This is defined as: $Z = \text{Density} \times \text{speed of sound}$. People like to compare this to the compressibility of a spring.

At a muscle-air interface, nearly 100% of incident intensity is reflected, ever noticed how air has some serious shadowing? *This is why gel must be used between the transducer and the skin to eliminate air pockets.*

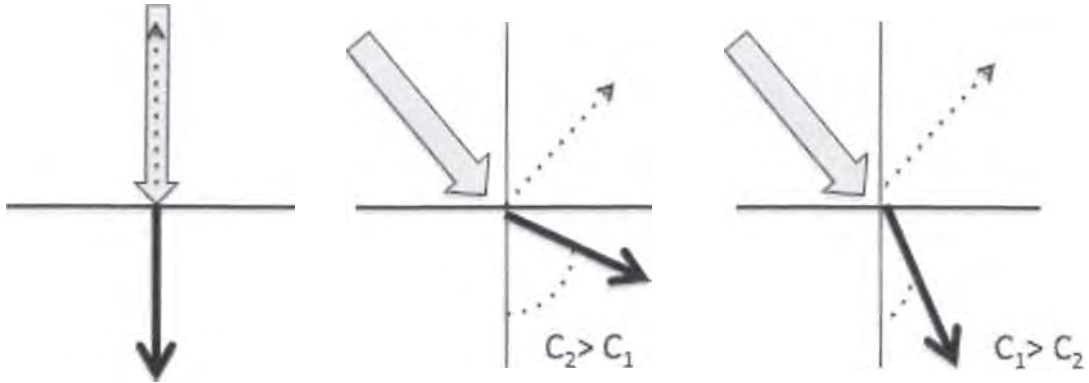
Refraction: This is the change in direction of transmitted ultrasound energy at a tissue boundary when the beam is not perpendicular to said boundary. Remember the frequency doesn't change, but the speed might.

What influences refraction ? Two Things: (1) Speed Change - which is based on tissue compression, and (2) the Angle of Incidence.

This was described by some dude named Snell:

$$\frac{\sin \text{Angle 1}}{\sin \text{Angle 2}} = \frac{\text{Speed 2}}{\text{Speed 1}}$$

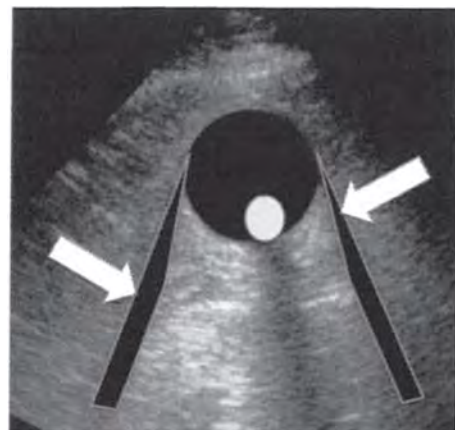
No refraction occurs if the sound is the same in the two media or with perpendicular incidence.



If it hits straight on, part of the beam will bounce straight back and part of it goes straight through. If it strikes at an angle, part will be reflected and the other part will be refracted - with the severity of this refraction depending on the speed difference of the two media.

Refraction is the cause of the shadows seen at the edges of a fluid filled structure (most commonly the gallbladder), as sound passes from tissue to fluid - altered from it's original path.

Total Reflection: It is possible to have complete reflection if the speed difference, and angle of incidence is great enough (exceeds the "Critical Angle").



Scattering: There are two main categories of reflectors:

- Specular (smooth) - The reflector dimensions are much larger than the wavelength of the incident.
**Strength of reflection is highly angle dependent.*
- Non-specular (diffuse) - The scattering surfaces are about the size of a wavelength or smaller. **Angle has no effect on strength*

With higher frequency ultrasound beams, the wavelength becomes smaller and the boundary no longer appears smooth from the perspective of the small wavelength. In this case, returning echoes are diffusely scattered leaving only a small fraction of the incident intensity returning to the source. Truly smooth reflections are relatively independent of frequency.

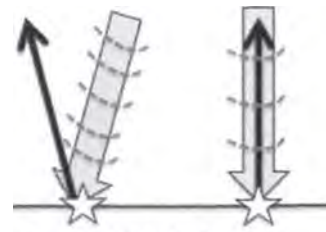
In other words: High Frequency = Small wavelength =
Surfaces appear more rough = More Scatter. So Higher
Frequency = More Scatter.

Differences in Scatter Amplitude: The difference in amplitude of the returning echoes from one region to another corresponds with brightness changes on the ultrasound display (essentially the number of scatterers per unit volume).

- Hyperechoic = High Scatter Amplitude - relative to average background
- Hypoechoic = Lower Scatter Amplitude - relative to average background

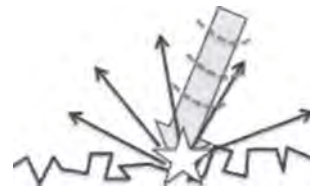
Absorption: Sound energy gets turned into heat. This increases with frequency.

“Attenuation” is the loss of intensity of the ultrasound beam from both absorption and scattering in the medium. The degree of attenuation varies widely depending on the type of tissue involved. To quantify the degree of attenuation, people who like math decided to express it in dB as proportional to frequency.



Smooth Reflectors:

*will reflect depending on
the angle of incidence*



Non-Specular:

*Will scatter everywhere
- angle doesn't matter*

If you are forced into a math problem, the rule of thumb for "soft tissue" is 0.5 dB per cm per MHz or 0.5 (dB/cm)/MHz.

It is proportional:

- A 2-MHz ultrasound beam will have twice the attenuation of a 1 -MHz beam;
- A 10-MHz beam will have ten times the attenuation per unit distance.

It is logarithmic:

- The beam intensity is exponentially attenuated with distance.

Testable Point: Half value thickness (HVT): This is the thickness of tissue necessary to attenuate the incident intensity by 50%, which is equal to a 3-dB reduction in intensity. As the frequency increases, the HVT decreases.

Think it Through: You always use the high frequency probe for superficial stuff right? This is why. The higher the frequency the more the sound gets attenuated, so the depth sucks. If you need to see a deeper structure then you have to use a lower frequency probe.

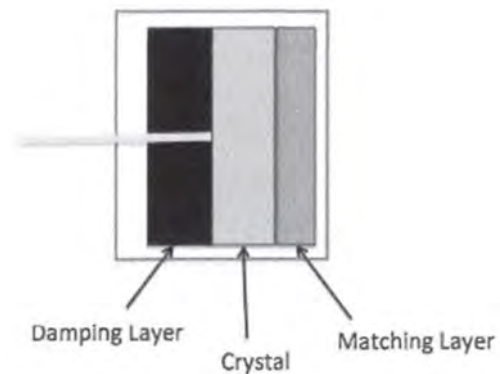
Review Time	
What determines the strength of the echoes?	Angle and Impedance
What is Impedance?	The degree of stiffness in a tissue. The differences in tissue impedance (stiffness) determines the strength of surface reflection.
What is the unit used for impedance?	The Rayl
The speed of sound is assumed to be?	1540 m/s
Attenuation is increased with?	Higher frequency ultrasound waves

Ultrasound Transducers and Related Trivia

Ultrasound is produced and received with a transducer. The device works by taking electricity and running it through a crystal (which vibrates) for conversion into mechanical energy (ultrasound waves). On the way back, the waves (mechanical energy) vibrate the crystal turning back into electricity so they can be recorded. Or something like that... close enough for what you need to know.

Back in the stone ages these things had a single element resonance crystal, now they have a broadband transducer array of hundreds of individual elements.

Piezoelectric Materials: This is a crystal (or ceramic) and is the functional component of the transducer. It could be quartz, but is usually lead-zinc-titanate (PZT). The magic to the material is a well-defined molecular arrangement of electrical dipoles. When mechanically compressed their normally organized alignment gets disturbed from equilibrium; this can be measured and recorded.



Resonance Transducers: These are made to operate in a “resonance” mode, where short durations of voltage (usually 150 V) are applied, causing the PZT to vibrate at a natural resonance frequency.

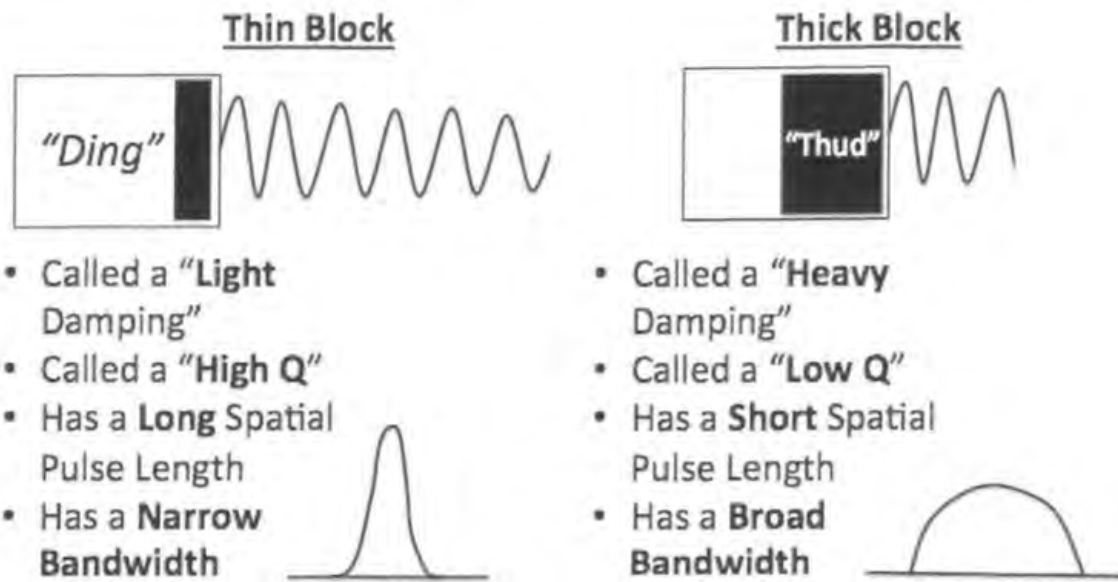
High Yield Trivia: The operating frequency is determined from the speed of sound in and the thickness of the piezoelectric material. The only way to change frequency is to change the probe. * *Wavelength changes to accommodate changing velocity in different media.*

High Yield Trivia: *The thickness of the transducer is equal to $\lambda/2$ the wavelength.* Lower frequency is seen with thicker crystals and higher frequency is seen with thinner crystals.

Dampening Block: This thing sits behind the crystal and absorbs the backward directed US energy. It also dampens the transducer vibration to create a pulse with a short spatial pulse length - needed to preserve detail along the beam axis (axial resolution). The process of dampening introduces a broadband frequency spectrum.

What you need to know about dampening blocks:

The differences in a thin block and a fat block easily lend themselves to multiple choice questions.



High Yield Trivia:

- **Low Damping (high Q)** - Narrow Bandwidth - *For Doppler, to preserve velocity information.*
- **Heavy Damping (low Q)** - Broad Bandwidth - **Gives you high spatial (axial) resolution** *fewer interference effects and therefore more uniformity*

Matching Layer - The matching layer gives the transducer an interface between the transducer element and the tissue. The key testable point is that it - **minimizes the acoustic impedance differences between the transducer and the patient.** It's made of stuff that has an acoustic impedance intermediate to soft tissue and the transducer material.

Testable Point: *The optimal matching layer thickness is equal to 1/4th the wavelength.*

Transducer Arrays

You can have Linear (which include curved) or Phased Arrays. Linear arrays are sequential, where as phased arrays are “activation / reactivation” types.

Linear	Phased
256-512 Elements	64-128 Elements
Large	Small
Simultaneous Firing of small group of adjacent elements (20ish)	Elements are activated sequentially
A rectangular field of view is produced (<i>trapezoidal with curved</i>).	Time delays in electrical activation can make it possible to steer and focus, without moving the probe

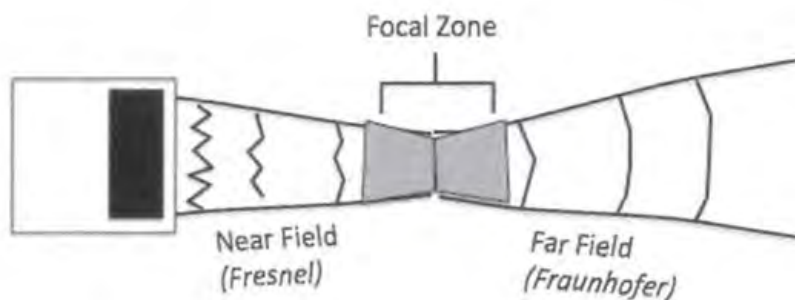
Why is a curved array curved? - Scan lines diverge deeper into the image. This gives you a wider field of view for deeper structures. Used in ABD imaging and OB.

Linear - This type of probe fires all elements simultaneously. This means the width of the transducer is equal to the width of the individual elements.

Phased - This type of probe fires elements at different times. The individual waves firing times can be changed to cause constructive and destructive wave summations - this steers and focuses the beam.

Beam Properties

There are two distinct beam components: a converging beam, which narrows out to a distance determined by the geometry and frequency of the transducer (near field), and a diverging beam (far field).



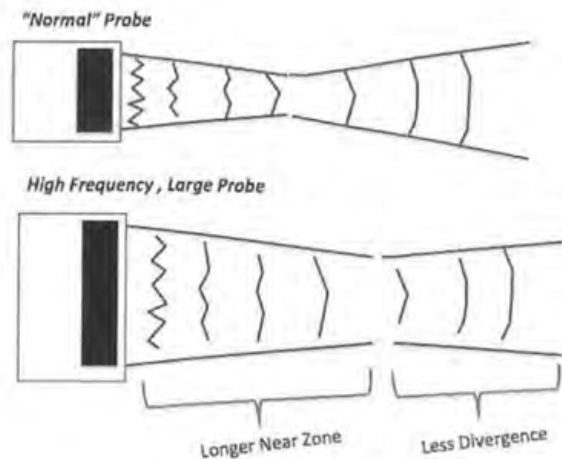
The Near Field (Fresnel Zone): The convergence of the near field occurs because of the multiple constructive and destructive interference patterns.

Beam intensity varies from: MAX-to-mm-to-MAX in a converging beam.

*The near field length is **dependent on:***
Transducer Frequency and Transducer Diameter.

Testable Point:

- Higher Transducer Frequency = Longer Near Field
- Larger Diameter Element = Longer Near Field



The Far Field (Fraunhofer Zone) - This is where the beam diverges. As the beam diverges the ability to distinguish two objects close to one another is reduced. The divergence is less with a high frequency big probe and more with a low frequency small probe. Ultrasound intensity in the far field gradually decreases with distance.

Intensity - The intensity of the beam is the power (measured in watts) flowing through a unit area. The power of the beam is not uniform along its length or width. This is caused by a variety of factors.

- The beam lacks clearly defined edges and the intensity will decrease from the center ➤ outward - sometimes called "beam spread"
- The maximum sound pressure is always found along the acoustic axis (centerline)
- Divergence of the beam in the far field causes the power to be spread over a larger area.
- Interference occurs from numerous point sources interacting in the near field. *Less with a broadband transducer

Focal Zone - The point at which the beam is at its narrowest and the area of maximum intensity. You get your best echoes here and you get your best lateral resolution here.

"Transmit Focusing" - There are several ways to do this, with the idea being to converge the beam (narrowing it) on the area you want to look at. Understand that the focal distance is a function of the transducer diameter (or width of group of simultaneously fired elements), the presence of any acoustic lenses and the center operating frequency.

- Shallow Focus* - achieved by firing outer transducers in the array before the inner transducers in a symmetrical pattern

- Longer Focus* - achieved by reducing the delay time differences among the transducer elements. This results in more distal beam convergence

- Multiple* - Multiple focal zones can be created by repeatedly acquiring data over the same volume, but with changes in the phase timing of the array elements. Because of the fact that each focal zone requires independent set of pulses, increasing the number of focal zones will decrease the frame rate and temporal resolution.

"Receive Focusing" - When using a phased array transducer, all the echoes received by the individual transducer elements are added together to create the signal from a given depth. Echoes received at the perimeter of the array travel from a longer distance than those received at the center of the array. To correct for this, the signals from individual transducer elements must be rephased to avoid a loss of resolution when the individual signals are brought together into an image.

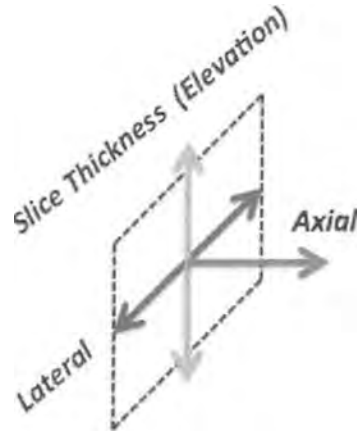
Time Gain Compensation (TGC) - This is the button you push to make the image look better. What it actually does is progressively increase the amount of amplification applied with depth. The man who lives in the ultrasound machine ("computer" I call it), is trying to make the top and bottom uniform. This used to require manual manipulation of buttons, but it is now automated.

What can you do to make this image better? - Answer is probably hit the TGC button.

Dimensions -

There are three dimensions in US:

- Axial
- Lateral
- Elevation (Slice Thickness)



Axial Resolution: The ability to tell two closely spaced objects apart in the direction of the beam. For optimal axial resolution you need the returning echoes to be separate and not overlap. The minimum required separation between two reflectors is $\frac{1}{2}$ the spatial pulse length (SPL), otherwise the returning echoes will overlap.

What is this "Spatial pulse length" ? This is the number of cycles emitted per pulse by the transducer multiplied by the wavelength.

Objects closer than $\frac{1}{2}$ Spatial Pulse Length will NOT be resolved.

Lateral Resolution: The ability of the system to resolve objects in a direction perpendicular to the beam direction. If you want to tell things apart, they must fit into separate beams. The thinner the beam, the more likely that each will fall into a separate beam. You can reason, that improving lateral resolution has a lot to do with making your beam thinner. The beam is most skinny at the end of the near field (at the focal zone). Lateral resolution is worst in areas close to and far from the transducer surface (away from the focal zone).

Is lateral resolution constant at different depths? No, Since the beam diameter varies with the distance from the transducer in the near and far field, the lateral resolution is depth dependent. The best lateral resolution occurs at the focal zone (the interface of the near field and far field interface). Lateral resolution worsens in the deeper field.

Is axial resolution constant at different depths? - Yep

Will cranking up the gain help? No - Gain widens the beam. You want a nice narrow beam. So, try and use the minimal necessary gain.

Elevation Resolution (Slice Thickness) - It's the same as lateral resolution but is measured in the plane orthogonal to the image plane. This is usually the worst measure of resolution. It depends on the transducer element height. This type of resolution is why you can get volume averaging of acoustic details in regions that are close to the transducer and in the far field.

Improving Axial Resolution	Improving Lateral Resolution	Improving Elevation Resolution
Shorter Pulses	Narrowing the beam in the proximal field (adding an acoustic lens). Minimal necessary gain (gain widens the beam).	Use a fixed focal length across the entire surface of the array (downside is partial volume effects)
Greater Damping (shorter pulses)	Phased array with multiple focal zones	Minimize slice thickness - done by phase excitation of the outer to inner arrays
Higher Frequency (shorter wavelength)	Increasing the "line density" or lines per cm.	

Dependent On	
Axial Resolution	Spatial Pulse Length
Lateral Resolution	Transducer Element Width
Elevation Resolution	Transducer Element Height

Artifacts:

Ultrasound display equipment relies on multiple assumptions to assign the location and intensity of each received echo. When these assumptions are violated you get an artifact.

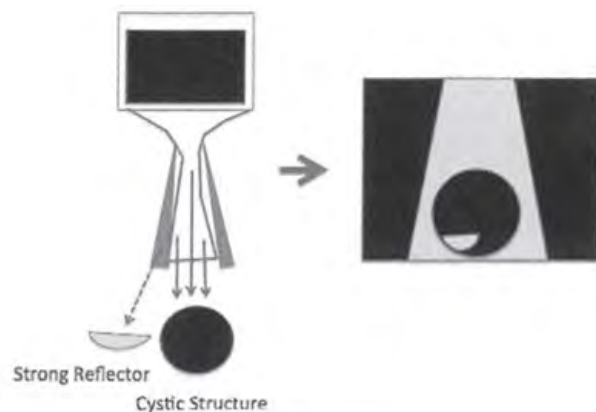
Assumptions:

- All echoes originate from within the main beam
- All echoes return to the transducer after a single reflection
- The amount of time an echo takes to return to the probe directly reflects on the object depth.
- The speed of sound in human tissue is constant - 1540 m/s
- The sound beam and its echo travel in a straight path
- Acoustic energy is uniformly attenuated

Beam Related Artifacts:

As stated above, there is an assumption that the detected echoes originate from within the main US beam.

Side Lobe Artifact: The normal US beam looks like a bowtie, with a central main beam and off axis low energy beams on the side. These off axis beams are the “side lobes.” You get these side lobes from radial expansion of piezoelectric crystals, which *happens more in linear arrays*. If you have a strong enough reflector it could bounce back this low energy side lobe, which will be received by the transducer. This will violate the assumption that echoes originate from the main beam - and it will be incorrectly placed as if it did. This artifact is typically seen when the incorrectly placed echoes overlap an anechoic structure (Bladder or GB). Some people refer to this as “pseudo sludge” when it’s seen over the gallbladder.



Testable Point: Side lobe energy is seen more with linear array transducers.

Beam Width Artifact: The US beam first exits the transducer with the same width as the transducer. It then narrows to the focal zone and then begins to diverge in the far field. The beam will eventually diverge out past the original margins of the transducer. If this diverged beam encounters a strong reflector, it could send a signal back - which will be assumed to be from the main beam (within the normal width of the transducer) and will be erroneously displaced as such.

The *bladder is the classic place to show this*, with peripheral echoes. A good “next step” question would be to show this artifact and have you improve it by (1) adjusting the focal zone to the level of interest and (2) *placing the transducer at the center of the image*.

Artifacts Associated with Multiple Echoes:

These artifacts violate the assumption that an echo returns to the transducer after a single reflection and that the depth of the object is related to the time for the round trip.

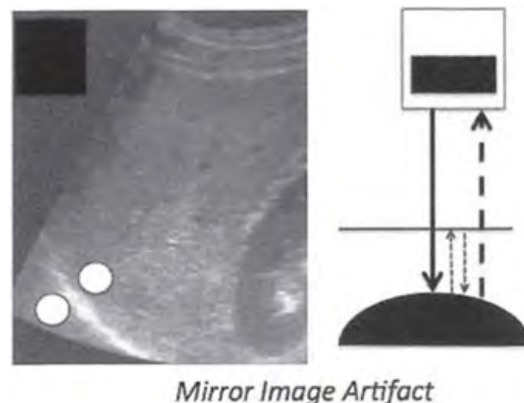
Reverberation Artifact: If the sound wave encounters two parallel highly reflective surfaces, the echoes generated from a primary ultrasound beam may be repeatedly reflected back and forth (like a game of pong) before eventually returning to the transducer for detection. This is recorded and displayed as multiple echoes, with the echoes that return after a single reflection being displayed appropriately and sequential echoes (which take longer to return to the transducer), erroneously placed at an increased distance from the transducer. This looks like multiple equidistantly spaced linear reflections.

Comet Tail Artifact: A form of reverberation. This time our two parallel highly reflective surfaces are closer together which means the sequential echoes are closely spaced. The space between them may be less than $\frac{1}{2}$ the spatial pulse length (SPL) - which as mentioned above was the minimal distance needed for axial resolution. As a result, the displayed echoes will look like a triangle, instead of linear lines.

Why a triangle and not a square ? The later echoes get attenuated and have decreased amplitude. This decreased amplitude is manifested on the display as decreased width. So you get a tapering triangle (or comet tail).

Ring Down Artifact: The mechanism for this one is slightly different. Instead of encountering two parallel highly reflective surfaces, our sound wave encounters fluid trapped between a tetrahedron of air bubbles. The vibrations create a nearly continuous sound wave transmitted back to the probe. You see this as a line or series of parallel bands extending **posterior to a collection of gas**.

Mirror Image Artifact: Like other artifact in this category, this is created by the false assumption that an echo returns to the transducer after a single reflection. In this situation the ultrasound beam passes through a highly reflective surface, then gets repeatedly reflected between the back side of the reflector and the adjacent structure. This is displayed as a duplication equidistant from but deep to the strongly reflective interface.



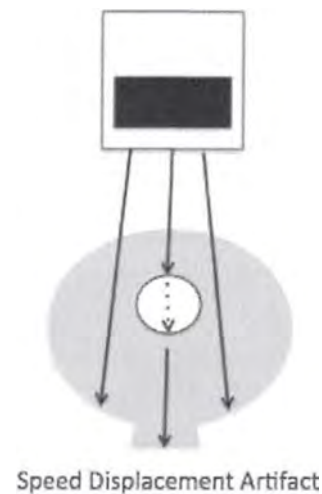
The classic location is along the liver / lung interface as shown - with liver parenchyma where you should have lung.

Artifacts from Multiple Echoes		
Reverberation	Two parallel highly reflective surfaces -	Multiple equidistantly spaced linear reflections.
Comet Tail	Two parallel highly reflective surfaces - closer together ($< 1/2$ SPL)	Triangle (comet) shaped
Ring Down Artifact	Fluid trapped between a tetrahedron of air bubbles	Parallel band extending posterior to a collection of gas
Mirror Image	Trapped behind a strong reflector	This is almost always shown with the liver on lung.

Artifacts Related to Velocity Errors:

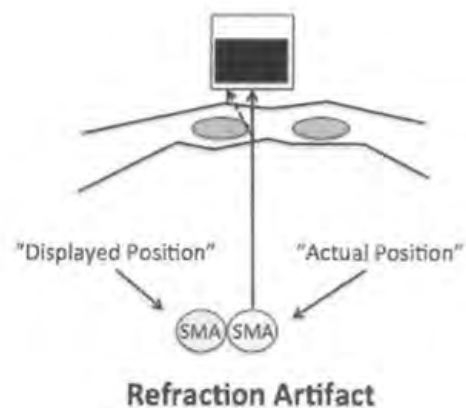
The assumption is that the speed of sound is 1540 m/sec in human tissue. Sometimes the beam encounters media (air, fluid, fat, bone) where this doesn't hold up exactly. If it travels slower- it takes longer to return and the machine thinks the depth is more. If it travels faster, it comes back to the monitor faster and the machine thinks the depth is less.

Speed Displacement Artifact: The speed of sound slows down in fat, relative to liver. This means the beam takes longer to return and is perceived by the machine as being further away. This creates the appearance of a discontinuous and focally displaced liver border.



Refraction Artifact: As discussed earlier in the US section, speed difference in tissues causes refraction (as described by Snell). This change in the direction of the sound wave violates the idea that the beam is going straight and can cause three things: (1) the object can appear wider than it actually is, (2) object can be misplaced to the side of the returning echo, or (3) object can appear duplicated.

The classic look (and location) for this is deep to the rectus muscles and midline fat. The so called “*duplicated SMA*,” occurs secondary to symmetry of the geometry. The SMA will become a normal single vessel if you move the transducer to the side (removes symmetry of refraction).



Artifacts Related to Attenuation Errors:

Ultrasound beams are attenuated as they travel through the body (from scatter and absorption). The longer the echo travels the more it will be attenuated (relative to an echo which traveled a shorter distance).

“Compensation Amplification ” - This is a type of processing ultrasound machines use on echoes that take longer to return to the transducer. Essentially, the echoes that return later are amplified more than echoes that return earlier. The end result is an image that appears more uniform in the deep field.

Shadowing - If the ultrasound beam runs into a material that attenuates sound to a larger degree than the surrounding tissue, the strength of the beam distal to this structure appears weaker (darker) than in the surrounding field.

Increased Through Transmission - This is the opposite of shadowing. Instead the beam runs into material that attenuates sound less than the surrounding tissue, the strength of the beam distal to the structure appears stronger (brighter) than the surrounding field.

Modes / Misc

A Mode, B Mode, M Mode

A Mode - “A” stands for amplitude. This is of historical context - and only used now by optho to take eye measurements. This gives you processed information from the receiver versus time. An echoe’s return from tissue boundaries and reflectors creates a digital signal proportional to echo. “A”mplitude is produced as a function of time.

B Mode - “B” stands for brightness. Basically this is the conversion of A line information to brightness-modulated dots on a display. There is a proportional relationship of brightness to the echo signal amplitude.

M Mode - “M” stands for motion. Essentially, B-mode information is used to display the echoes from a moving organ (like heart valves) from a fixed transducer and beam position on the patient.

Harmonics

As the ultrasound beam travels into the patient's tissues the transmitted pulse is progressively distorted. However this occurs primarily in the central zone of the main beam where the intensity is high. A distorted pulse will give rise to distorted echoes and these have significant energy at harmonic frequencies. Tissue harmonics are made using the second harmonic component of the echo signal, with the fundamental frequency excluded. Undistorted echoes coming from lower intensity areas (fringes of the beam, side lobes, superficial tissues) are not seen.

Advantages	Disadvantages
Improved lateral spatial resolution	Being at a high frequency, the second harmonic attenuates far more rapidly than the fundamental, so the depth of penetration is reduced
Reduced Side Lobe Artifact	
Removal of Multiple Reverberation Artifact - from adjacent anatomy	The processing used to remove the fundamental frequency generally requires that two transmitted pulses are used for each line of sight in the image. This reduces the frame rate by a factor of two.
Cysts look clearer.	Hypoechoic masses look like cysts

Mammo Ultrasound - Special Topic

Compound Imaging - Having the electronic steering of the ultrasound beams from the transducer image an object in multiple different directions. This will sharpen the edges and cause loss of posterior shadowing (can make a cyst look solid).

Compare and contrast normal settings, harmonics, and compound imaging of a hypoechoic mass in mammo.



Normal Settings

- Hypoechoic Mass
- Blurry Margins
- Some Posterior Shadowing

Harmonics

- Looks more anechoic
- Blurry Margins

Compound Imaging

- Hypoechoic Mass
- Sharp Margins
- Lost Posterior Shadowing

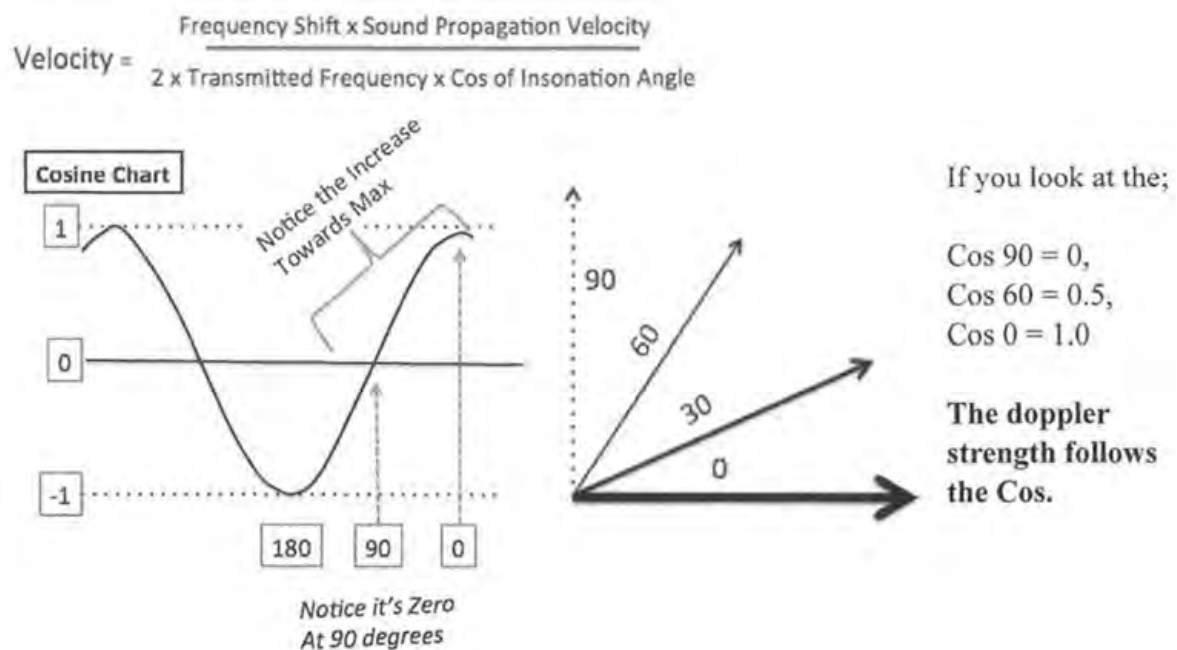
Doppler

Doppler Shift: When sound is reflected from a moving object and the frequency of the reflected sound changes - PhDs call that a “Doppler Shift.” With some math, this change in frequency can be used to determine the speed and direction of blood.

Doppler Angle: In an ideal world, the sample volume would be placed in the mid-part of the lumen, with an angle of incidence parallel to the vessel. If this angle is placed incorrectly the velocity calculation will be incorrect (especially if the angle is greater than 60).

The angle should be between 30- 60.

Why less than 60? Because of the formula for calculating it. Notice that the cosign of the angle is on the bottom.



Looks like zero is best. So why more than 30? - Angles less than 20 degrees cause refraction and loss of signal. Aliasing also becomes an issue.

Why Not Perpendicular ? - If you placed the probe perpendicular to the vessel, it can give a false impression of no flow (occlusion). The other thing it can do is create a mirror image.

Pulsed Wave (Spectral) Doppler- This utilizes a single transducer for both reception and transmission. Blood flow velocity varies yielding a spectrum of doppler shifts instead of a single frequency. You can obtain the direction of blood flow (plotted above and below the baseline) and velocity.

Color Doppler: This uses the gray scale image with a superimposed color blood flow image. Gives the direction of flow, this time with colors (red and blue). Color intensity varies depending on the flow intensity (things holding still are gray). This type of doppler obtains samples of each pixel multiple times then displays the average shift.

Testable Trivia: The spatial resolution is less compared to gray scale imaging (although smaller vessels are better seen).

Testable Trivia: The doppler angle is not as important since the information is semi-quantitative.

Power Doppler: Power doppler is different because it gives you a very sensitive look for the presence of flow without information on direction. You still get color but instead each pixel registers the total number of “frequency shifts”.

Testable Trivia: Power Doppler does NOT exhibit aliasing (*both color and spectr-al can*).

Testable Trivia: Power Doppler has NO dependence on the Doppler Angle - *you can measure doppler totally perpendicular to the vessel.*

Testable Trivia: Power Doppler is extremely sensitive to flow (*even very slow flow*)

Doppler Artifacts:

Aliasing - Super high velocities are displayed as low (negative). On a spectral system it looks like the spectrum is cut off and “wrapped around the baseline”, reappearing on the opposite end.

Testable Trivia: This artifact occurs when the doppler shift is greater than a threshold called the “Nyquist frequency”

$$\text{Nyquist limit (kHz)} = 1/2 \times \text{pulse repetition frequency (PRF)}$$

Example: A frequency shift of 3.5 kHz requires a PRF of 7 kHz to avoid aliasing.

Aliasing can be reduced or eliminated by:

- Decreasing Doppler shift - by either using a lower frequency transducer or using a doppler angle closer to 90
- **Increasing the Pulse Repetition Frequency** (which will increase your Nyquist) or selecting a sample volume at a lesser depth or **increasing the scale.**

Tissue Vibration: This occurs secondary to turbulent blood flow. The doppler shows a mixture of red and blue colors. The classic example is a **A-V fistula in a kidney post biopsy**.

Minor Image: Just like the gray scale version, this occurs secondary to a vessel adjacent to a highly reflective surface, such as the lung. This results in the duplication of the structure being evaluated. The classic locations are the subdiaphragmatic region of the liver and the supraclavicular region.

Twinkle Artifact: This occurs behind strongly reflecting surfaces such as calcifications. It manifests as a noisy spectrum with rapid fluctuation of red and blue colors.

Testable Trivia: This artifact has a greater sensitivity for detection of small stones than acoustic shadowing.

Testable Trivia: This is highly dependent on machine settings and how round the reflecting surface is (more rough = more twinkle).

Pseudoflow (Pseudoblood) Artifact: Things that move like blood but aren't. The classic example is **ureteral jets**.

Flash Artifact: This is a burst of color filling the screen. It's secondary to transducer or patient motion. The classic example is the **"fetal kick!"**

Color Bleed - This looks like color extending beyond the vessel wall. It can decrease sensitivity to thrombus or stenosis.

Making it better: Decrease the color gain.

Power and Gain - Some Quick Points:

- Increasing power increases your penetration depth.
- Gain only changes the brightness on the imaging monitor, but has no affect on the actual ultrasound output.
- If you crank up the power too much, you can create an artifactual image.

Safety

Two acoustic output parameters are used as indicators of the potential for biologic effect.

- Thermal Index* - this is the maximum temperature rise in tissue secondary to energy absorption. This is based on a homogeneous tissue model with certain instrument parameters.
- Mechanical Index* - this is how likely it is that cavitation will occur considering peak rarefaction pressure and frequency. This index is the indicator of mechanical bioeffects (streaming and cavitation). **This matters the most with contrast enhanced US.**

Cavitation - Sonically generated activity in compressible bodies composed of gas and/or vapor. This can be sub-classified as either stable or transient.

•**Stable Cavitation** - Micro bubbles are already present in the media. They expand and contract as the waves cycle (responding to pressure). This occurs at low and intermediate ultrasound intensities (as used clinically). The MI is an estimate for producing cavitation.

•**Transient Cavitation** - “The violent one” - Bubble oscillations become so large that the bubbles collapse. This collapse results in shock waves rippling through tissue planes causing tissue damage.

Testable Trivia: The rate of energy absorption increases with frequency. Eventually the rise in temperature slows secondary to conduction and perfusion with an eventual steady state.

Testable Trivia: Thermal induced damage is a threshold phenomenon (you get no tissue damage until a certain temperature is reached).

Testable Trivia: Cavitation is most likely to occur with low frequency and high pressure.

Testable Trivia: Spectral Doppler deposits more heat compared to gray-scale ultrasound.

Testable Trivia: Per the NCRP - a risk-benefit decision when the TI exceeds a value of 1.0 and the MI exceeds a value of 0.5.

Obstetrics:

The bottom line is ultrasound has been around for fucking ever, and there has never ever ever been a single case of a human fetus injured by it. Having said that, Radiologists are massive cowards so caution is still advised.

Since temperature rises are most likely at bone surfaces and adjacent soft tissues, a baby with increasing mineralization (2nd and 3rd trimester) has the theoretical risk of the possibility of heating sensitive tissues such as brain and spinal cord.

Testable Trivia: The Thermal Index for Bone “TIB” applies to an ultrasound beam passing through soft tissue then hitting bone (the scenario for 2nd and 3rd Trimester).

General Recommendations (not at all evidenced based) for 1st Trimester:

- Pulsed doppler (spectral, power, and color) should NOT be routinely used
- Keep the TI under 1.0 (some sources say 0.7).
- Scanning maternal uterine arteries with doppler is probably ok (as long as the fetus is outside the beam)

Thermal Index:

- Below 0.7 - for OB imaging
- Between 1.0 - 1.5 - US should NOT exceed 30 mins
- Between 2.5 - 3.0 - US should NOT exceed 1 min
- Greater than 3.0 - US should NOT be used.

You mark that frame an 8, and you're entering a world of pain

Section 8: Nukes

Nuclear medicine is unique in that instead of passing a beam of energy through the patient, you are injecting a tracer into the patient and observing a normal or abnormal distribution of that tracer.

Stability / Decay: Everyone wants to be happy. I want to be happy, you want to be happy, and even isotopes want to be happy. We seek it in different ways. Some of us incorrectly thought that going to medical school would make us happy. Isotopes seek happiness through stability. In general, what makes an isotope stable is a balanced number of protons and neutrons. The process of emitting energy or particles is an attempt to become more stable.

Some Vocab: “*Transmutation*” - A change in the number of protons, which by definition changes the element.

Subtypes of Transmutation:

- * *Alpha Decay* = This tends to occur in heavier unstable atoms. Alpha particles are basically Helium nuclei (2 protons, 2 neutrons). Alpha particles are slow and fat. They can't even penetrate a piece of paper, and are worthless for imaging.
- * *Beta Minus Decay* = **Seen with Neutron Excess.** A neutron is converted to a proton, then emits an electron (*beta particle*) and antineutrino. The range of these dudes is about 1cm, so they are also worthless for imaging. They do have the potential to harm DNA and form the basis of radionucleotide therapy with ^{32}P , ^{89}Sr , ^{90}Y , ^{131}I , and ^{153}Sm .

$\text{Neutron} \rightarrow \text{Proton} + \text{Electron} + \text{Antineutrino}$
- * *Beta Plus Decay* = **Seen with Proton Excess (Neutron Deficiency).** A proton is “transformed” into a neutron. (*Trivia to know=you need 1.02MeV for this to occur*) A positron (*beta particle*) is then emitted which travels a short distance before colliding into a real electron and then destroying each other. The mutual destruction emits **two 511 keV photons which come out 180 degrees apart.** Beta plus and Electron capture both occur in the setting of proton excess (neutron deficiency) and therefore compete with each other.

* *Electron Capture* = Also **Seen with Proton Excess** (*Neutron Deficiency*). This occurs in the setting of insufficient energy (*remember beta plus needs 1.02MeV*). A proton eats (captures) an electron and then turns into a neutron. The neutron (*formerly a proton*) then burps (*emits*) characteristic radiation

Alpha Decay	Heavy Unstable Atoms	Lots of tissue damage
Beta (-)	Neutron Excess (<i>Proton Deficiency</i>)	Electron Emission Can Damage DNA (basis of Radionucleotide Therapy)
Beta (+) <i>-Rival Of Electron Capture</i>	Proton Excess (<i>Neutron Deficiency</i>) <i>Has at least 1.02MeV</i>	Positron Emission , leading to two 511 keV photons which fly 180 degrees apart
Electron Capture <i>-Rival Of Beta (+)</i>	Proton Excess (<i>Neutron Deficiency</i>) <i>Does NOT require 1.02MeV</i>	Leads to gamma emission (sometimes) and characteristic radiation, both of which may be used in imaging

High Yield Trivia: Particle emissions cause more problems than photon emissions. What do I mean problems? Even though Beta (-) and Beta (+) don't travel far, they can damage DNA. So plastic should be used to shield against them (NOT LEAD because it will create Bremsstrahlung x-rays). So just remember **B emission = Plastic Shield (NOT LEAD)**.

Rivals

- * *Isometric Transition* - **“The Good Guy”** Any process that gives off gamma radiation but Protons and Neutrons don't change.
- * *Internal Conversion* - **“The Bad Guy”** Excess Energy Exceeds Binding Energy so you get an Ejected Electron plus some characteristic radiation.

These two dudes compete with each other and their ratio is termed the "Alpha. " The lower the alpha the more useful radiation you get and the less harmful radiation you produce.

Low Alpha = Good.

Tc has a low Alpha.

More Vocab:

Metastable: If you have some delay between particle decay and gamma emission then you can call it

“Metastable.” The classic example (and probably only one you need to know) is the metastable Tc99.

142.7 KeV Tc99M (half life 6 hours)

140.5 KeV

I

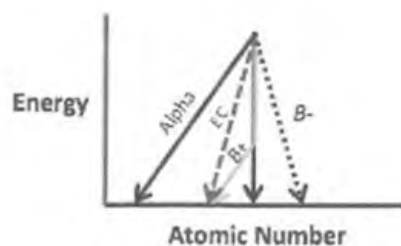
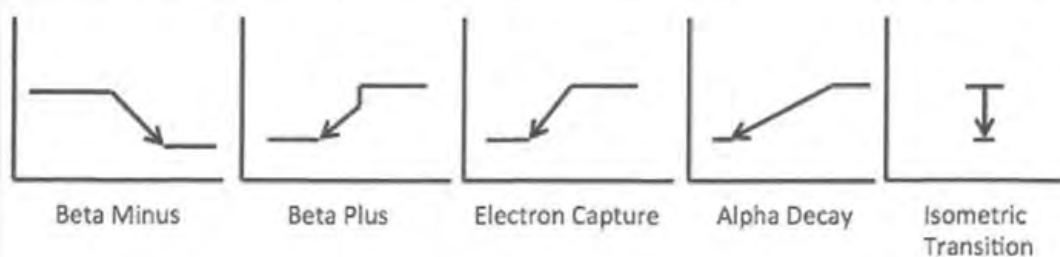
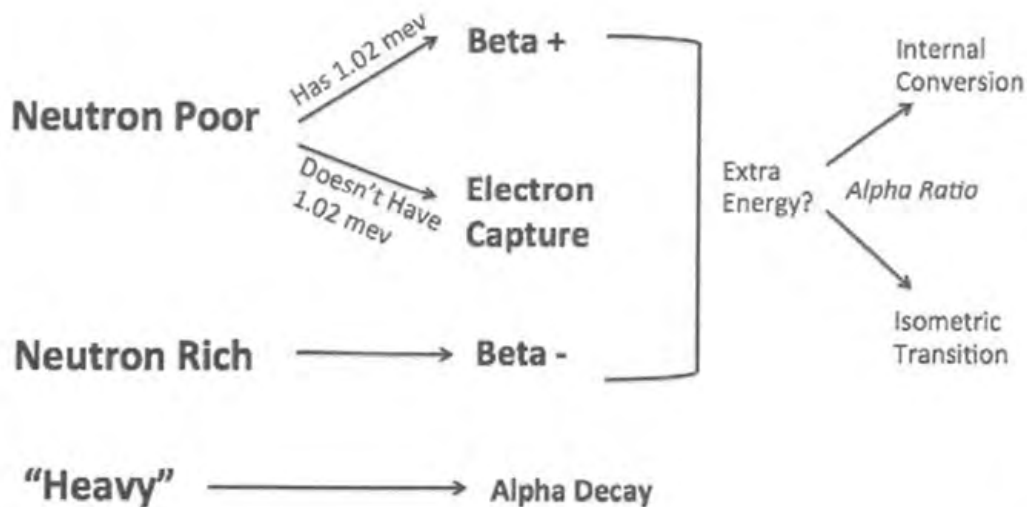
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0 KeV

Tc99 (half life million billion billion years)

Summary Chart:

Here are some classic diagrams that illustrate the different processes of decay.

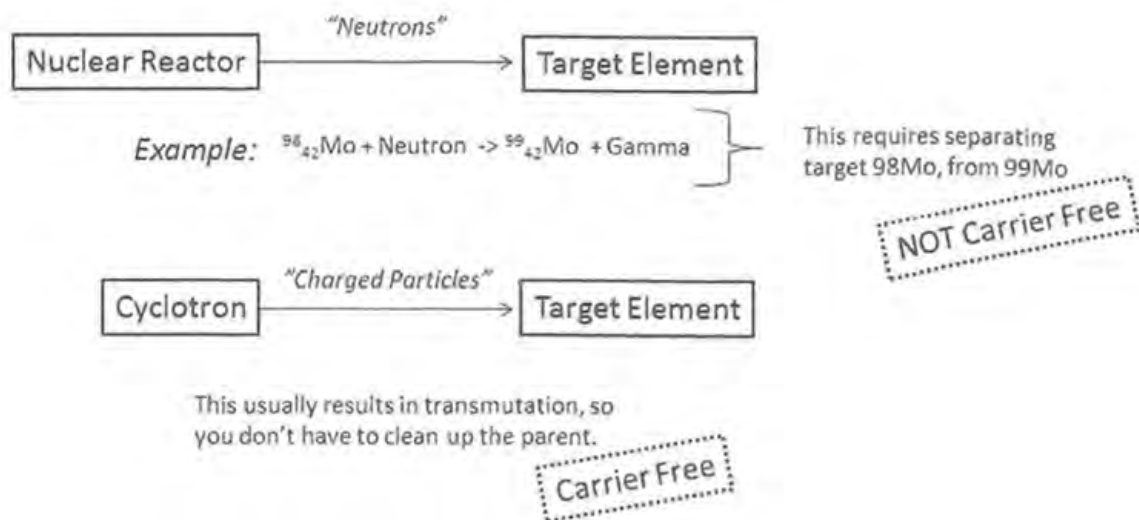


Production

There are two main ways you can create tracers, both of which alter the Neutron/Proton ratio to make them unstable:

- (1) Bombardment (in a nuclear reactor or cyclotron)
- (2) Fission

Bombardment: This is basically striking target elements with either neutrons (in a nuclear reactor), or with charged particles (alpha particles, protons, or deuterons) in a cyclotron. The downside to the nuclear reactor method is that you have to clean up the left over parent element. This is referred to as not being carrier free. The cyclotron has the advantage of producing elements via transmutation, therefore you don't have any parents to clean up "Carrier Free."



Fission: Neutrons are fired into large atoms (like Uranium and Plutonium) and split them into pieces. Basically just results in a bunch of random crap being made; Iodine-131, Xenon-133, Strontium-90m Molybdenum-99, Cesium-137. Obviously the desired isotope also has a bunch of fission products (contaminants) which have to be separated out - can be done with chemistry. These guys demonstrate a bunch of different decay methods.

Neutron Activation: Target atoms eat up neutrons to form a new isotope. These neutrons don't really even need to be accelerated. Because the products are isotopes of the target atoms they cannot easily be separated from each other. As a result these are NOT carrier free. These neutron rich products tend to decay via beta emission.

Trivia:

- *Neutron Bombardment and Nuclear Fission -> Elements with Neutron Excess (will decay via beta emission)*
- *Cyclotron -> Elements with Neutron Deficiency (will decay by electron capture or positron emission)*

<i>Neutron Bombardment</i>	<i>68-Ga, 82-Rb, 99m-Tc, 113m-In</i>
<i>Cyclotron</i>	<i>11-C, 13-N, 15-O, 18-F, 111-In, 123-I, 67-Ga, 201-Tl</i>
<i>Fission</i>	<i>99-Mo, 131-I, 133-Xe, 137-Cs</i>

Radioactive Decay

“**Activity**” is defined by the amount of disintegrations per second. Historically this was measured in Curie (Ci) which is 3.7×10^{10} disintegrations per second. **The new SI unit is the Becquerel (Bq), which is one disintegration per second.**

“**Specific Activity**” is defined as the activity per unit of mass (Bq/g). The longer the half-life, the lower its specific activity.

“**Half Life**” - The physical half-life. The amount of time necessary for a radionuclide to be reduced to half of its existing activity. In addition to **physical half life**, you have **biologic half life**. The biologic half life is basically how long it takes to shit or piss half the tracer out. The “**effective half life**” takes both of these things into consideration.

$$1/T_e = 1/\text{Physical} + 1/\text{Biologic}$$

Example: I-131 has a T_A -Biologic of 24 days

$$1/T_e = 1/8 + 1/24 = 1/6, \text{ so } 6 \text{ days}$$

Now having said that - if you have a large mismatch with biological and physical half life, effective half life basically becomes the short one. In other words, if you breath the Xe out in 15 seconds it doesn't matter what the physical half-life is. On the other hand, if your biological half-life is 10,000 years then you are 100% counting on physical half-life to clear it.

How long do you have to keep radioactive material? The general rule is 10 half lives.

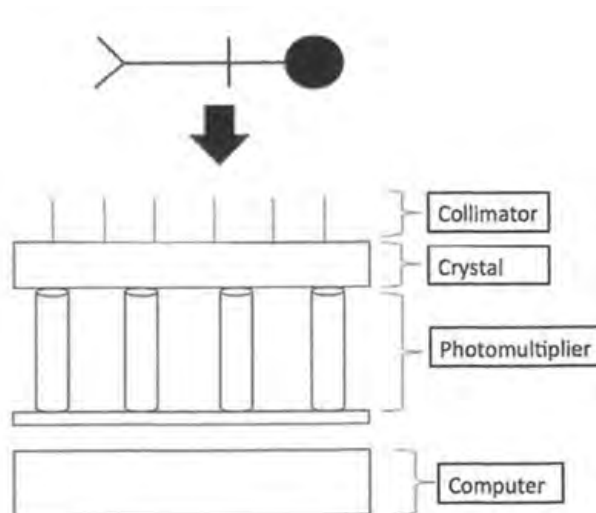
The Perfect Tracer:

- Ideally photons need to be emitted within a range that can be detected by a camera. Ideally between 100-200 keV.
- Ideally the tracer should have no particle emissions (*this increases the dose, without making the image better*).
- The half life needs to be long enough to be imaged, but not too long that you are getting extra dose.

How the Gamma Camera Works:

A gamma camera takes photons emitted from the radionuclide, turns it into a light pulse, and then takes that light pulse and makes some voltage. The voltage makes a picture.

Collimator: There are a lot of unwanted photons (background, off-axis, Compton scatter, etc...). The purpose of the collimator is to reduce scatter and allow for correct localization of radionuclide events. It works by discriminating based on direction of travel. It can NOT tell the difference in photon energy (that's done by the photon height analyzer).



Parallel Hole Collimator: “The Work Horse” This can be modified based on the energy of the tracer being used.

- * Low Energy (1-200keV)- Thinner Plates: ^{99m}Tc , ^{123}I , ^{133}Xe , ^{201}Tl
- * Medium Energy (200-400keV): ^{67}Ga , ^{111}In
- * High Energy (> 400keV) - Thicker Plates: ^{131}I (technically most energy peaks are medium)

•**Sensitivity vs Resolution: These two guys have an inverse relationship.** As one goes up the other goes down. A high sensitivity collimator will allow twice as many counts to be imaged but will degrade the spacial resolution. High sensitivity collimators are important with dynamic imaging (like the flow phase of a bone scan).

•**Effects of Distance on Sensitivity and Resolution: Distance has NO effect on Sensitivity** (increased distance reduce counts by inverse square, but the increased distance allows for a greater field of view. NO change occurs in the net counts). **Distance DOES affect Resolution** (septa are no longer able to eliminate photons from oblique angles as distance increases).

•**Septal Length (collimator depth):** Short septa give a crappy spatial resolution, but better sensitivity. Long septa give excellent spatial resolution, but crappy sensitivity (noisier image).

•**Hole Diameter:** Wide holes = Highly sensitive, low resolution. Narrow holes = low sensitivity, high resolution.

Parallel Hole Factor		
Septa Length	Long Septa: - Low Sensitivity (Noisy) - High Spatial Resolution	Short Septa: - High Sensitivity - Low Spatial Resolution
Hole Diameter	Wider Hole: - High Sensitivity - Low Resolution	Narrow Hole: - Low Sensitivity - High Resolution
Septa Thickness	Thick Septa: - Less Penetration - Less Available space for holes (Less Sensitivity)	Thin Septa - More Penetration (Blur) - More Available space for holes (More sensitivity)

In General:

- You want to use long thick septa + wide holes for high energy
- You want to use short thin septa + narrow holes for lower energy

Pinhole Collimator: Magnifies and inverts image. Used for thyroids and other small parts. It's usually cone shaped.

Magnification occurs at a ratio of: pinhole to detector " f " / pinhole to patient " b "

- If $F = B$ there is no magnification
- If $F > B$ = there is magnification
- If $F < B$ = object gets smaller.

In other words, if you move it far enough back it will make the image smaller. Magnification at the front is greater than the back (objects are 3D), this is why large objects get distorted and pinholes work best on small things. Sensitivity for pinhole cameras is garbage.

Converging Hole Collimator (Cone Beam): Holes are close together on the object side and far apart on the crystal side. **Magnifies WITHOUT inverting the image.**

Diverging Collimator: The opposite of converging. Holes are far apart on the object side, and close together on camera side. This **takes a large object and minimizes it**. The result is that you can image a large part of the body on a small crystal. So increased area, decreased sensitivity and resolution.

Collimator Type	
Parallel Hole	"The work horse." You want the collimator and detector as close as possible to the patient for the best spatial resolution (this is affected by distance). Sensitivity is NOT affected by distance.
Pinhole	Magnifies and inverts image - used for thyroids and other small parts. Large objects get distorted (front is magnified more than back). Sensitivity is garbage.
Converging	Magnifies without inverting
Diverging	Takes a large object and makes it small.

Scintillation Crystal- Once the photon emerges from the collimator, it impacts on the crystal. The crystal is made of sodium iodide doped with thallium. The crystal has the property that when struck with a photon it produces a pulse of light.

The thicker the crystal the less photons will be wasted (just pass through). On this same principal, thin crystals will let more photons through (without generating light). Energy of the photons themselves also plays a similar role. Higher energy photons will pass right through the crystal (lower ones won't). A thicker crystal is obviously better for high energy photons. So why not just make all the crystals super thick? Apparently you sacrifice spatial resolution with a thick crystal, because with a thinner crystal the photomultiplier tubes can sit closer to the event and more accurately localize it.

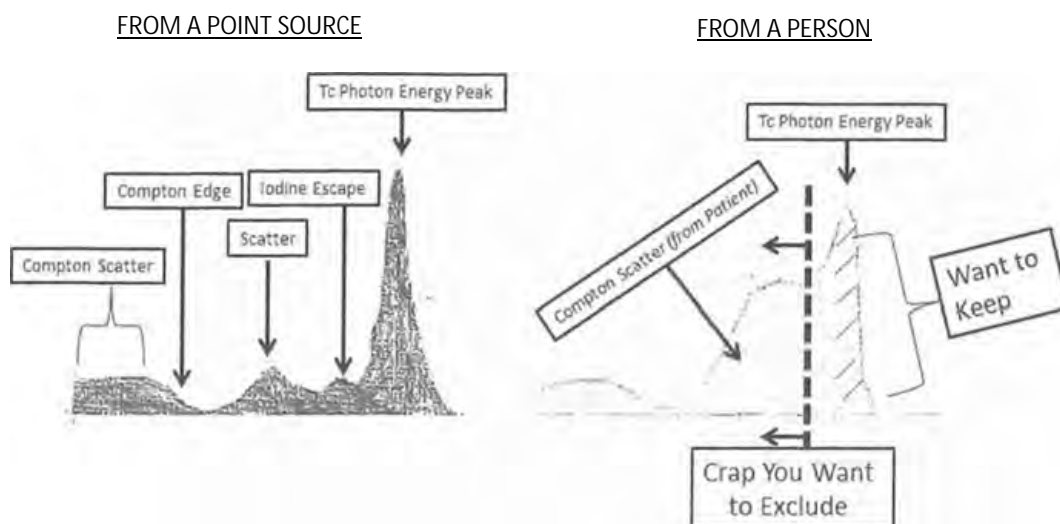
* *Thick crystal = Better Sensitivity, But Worse Spatial Resolution*

* *Thin crystal = Better Spatial Resolution, but Worse Sensitivity*

Photomultiplier Tubes (PMT) - The PMTs detect the light and convert it into an electric signal or measurable magnitude. The more PMTs you have, the more light you pick up and the greater the resolution. The PMTs record two things: (a) location - on x and y axis, and (b) signal intensity "Z." The X and Y coordinates go straight to the computer. The Signal Intensity (Z) goes to this gadget called a pulse height analyzer (discussed next).

Pulse Height Analyzer - The function of this thing is to discard background crap, and only look at the photons from the tracer you are looking for. The background crap can come from multiple sources (compton scatter, iodine escape from the crystal itself, backscatter).

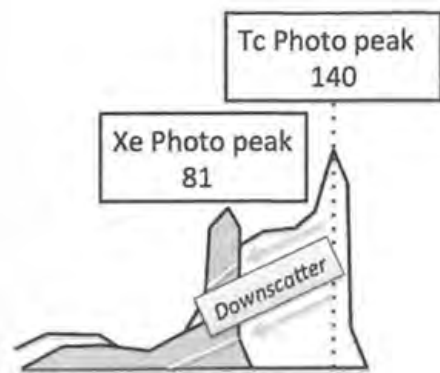
The texts make a point in distinguishing the difference of background from a point source versus background from a person. Basically you should know that the Compton scatter in a person is a lot closer to the energy you want to image, but really degrades the images.



You can set a window that will exclude all that crap higher or lower to the desired peak. Some cameras allow you to record multiple peaks (great for things like ^{67}Ga and ^{111}In which have multiple peaks or dual tracer studies).

Key Concept: “Downscatter”- There is a thing called downscatter, which is super super high yield. Essentially high energy photons can spill into the window of a low energy emitter, mainly resulting from Compton scatter effects.

Example 1: You are doing a V/Q scan using ^{133}Xe and $^{99\text{m}}\text{Tc}$. Xe has an energy of 81 keV, whereas Tc has an energy of 140keV. The Tc Compton scatter is gonna range down from about 135 to 90 (as in the above diagram “from a person”). So if you inject the Tc first and image that will turn out just fine. But when you give the patient the Xe they will still have the Tc on board so you will be getting Xe peaks at 81, with a bunch of Tc Compton scatter all over it. So you totally hosed yourself, because the Pulse Height Analyzer can’t tell what is Compton scatter from the Tc and what is signal from the Xe. The solution is to image with the Xe first, because the 81 keV and downstream Compton effects from the Xe would not affect the peak at Tc (140) at all. Hopefully that makes sense. Otherwise just remember, **LOWER PHOTON ENERGY TRACERS MUST BE USED FIRST.**



Example 2: If you are using $^{99\text{m}}\text{Tc}$ and ^{67}Ga for a bone scan of the spine looking for osteomyelitis. Gallium has a bunch of photopeaks (93, 184, 300, 393) and an effective half life of 50 hours. You have to give the Tc first and image, otherwise you’d end up waiting 50 hours.

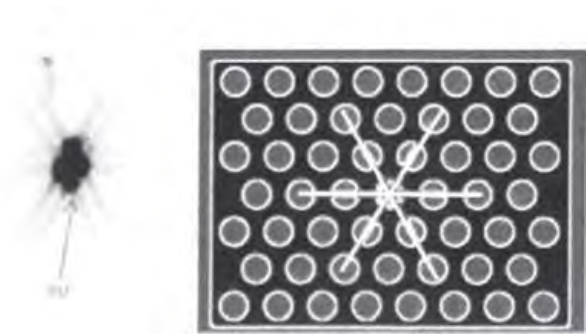
SPECT and Matrix:

Gamma camera imaging can be “static” or “dynamic.” When improved spatial resolution is needed “SPECT” can be performed by rotating a camera 180 or 360 degrees around the patient. The longer you image on SPECT the better it looks, but patients just can’t hold still forever - and movement degrades the images. Another thing to consider is matrix size.

Matrix size: Size matters in most things in life, matrix being no different. A matrix size of 128x128 has superior resolution to 64x64. However, a larger matrix size means longer acquisition time (which patients hate), and reduced count density per pixel (which impacts image contrast)

Star Artifact:

If you have very focal intense energy you can sometimes see a star artifact, **caused by septal penetration of the hexagonal collimator holes.** This is typically seen in the thyroid bed after a high therapeutic dose (using a medium energy collimator, instead of high).



Star Artifact - From Septal Penetration
**Seen if collimator holes are arranged in a hexagonal pattern*

Quality Control for Gamma Cameras

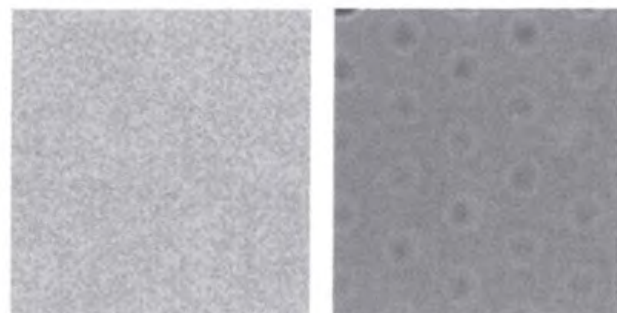
Possibly the most dreaded portion of radiology multiple choice exams (*right up there with safety, and mammo MQSA*). Daily, weekly, and quarterly tests are done on the camera (**BY A TECH! NOT A RADIOLOGIST!!!**) to make sure it's functioning properly. Four parameters are usually tested for: field uniformity, window setting, image linearity and spatial resolution, and Center of Rotation (COR).

Field Uniformity: So the photo multiplier tubes can have subtle variability in what voltage they assign to a given photon of light. Additionally, the crystal isn't totally uniform, and has subtle variations in thickness. For exam purposes, you want to try and keep these two things as uniform as possible. A 2%-5% non-uniformity is allowed (1% if SPECT).

The test is known as a **"Flood"**, and is used to see if the camera can produce a uniform image along the entire crystal surface. It's done two ways: (1) extrinsically (with a collimator), and (2) intrinsically (with NO collimator). You can use either a $Na^{99m}TcO_4$ source or a Co^{57} source to perform the flood. The **recommended counts for both extrinsic and intrinsic range somewhere between 5-10 Million** (*Siemens recommends 10 Million*).

- Extrinsic (WITH Collimator) - is done daily. This tests the collimator and crystals.
- Intrinsic (WITHOUT Collimator) - is done weekly.

A computer will check to make sure the field is uniform. You can also visually inspect the test images, to see if there is an obvious problem like a bunch of bullseyes, or a segmental region of failed PMTs.

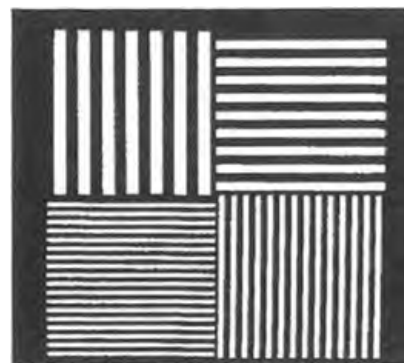


Normal
- "Flood" - For Field Uniformity

Bull's Eye
Appearance of Tubes Indicates
A Problem

Energy Window: The correct window needs to be used prior to each study, therefore this test **should be performed daily**. The most common approach to testing this is to use a symmetric window centered at the peak energy used in the imaging test. The source can be a syringe, a vial, or if absolutely necessary the patient. For Tc you would use a 20% window centered at 140keV.

Image Linearity and Spatial Resolution: Lead bar phantoms with parallel lines, placed between the collimator and a ^{57}Co sheet, are used to test image resolution and linearity. This only needs to be **done weekly**. Resolution is defined by the ability to differentiate between two distinct points (can you tell the bars are separate). Linearity is tested by looking to see if all the bars are straight (some distortion at the edges is ok). The bars should NOT look wavy.



Bar Phantom
– Lines should be straight (not wavy)

Center of Rotation: Gamma cameras that are used for SPECT have to be routinely monitored for alignment offset at the COR (Center of Rotation). The test is **done with 5** small ^{99m}Tc **point sources** along the axis of rotation. The **axis should be straight**, with minimal deviation. This is **performed weekly**.

Lead Aprons / Film Badges / Ring Badges

Why don't nuclear medicine techs wear lead?

Two reasons: (1) Thin lead doesn't stop gamma rays. To stop them it would have to be so heavy you couldn't move. (2) When high energy gamma rays collide with a dense material that is not thick enough to stop them (lead apron) they rapidly slow down and lose energy which turns them into deeply penetrating Bremsstrahlung x-rays - which actually makes the dose worse.

Correct Positioning on the Film Badge and Ring Badge

- * **Film Badge:** Should be worn on the collar at the chest / neck level
- * **Ring Badge:** Worn on **dominant hand, index finger, label in towards source**
(usually this means towards the palm), **under a glove (to avoid contamination)**

Safety:

This is also an excruciating topic for Radiologists to learn about. As previously stated nukes is full of random trivia, and let's be honest academic nuclear medicine tends to attract eccentric personalities... and they are writing the questions.

Instruments:

- **Geiger -Muller Counter** - this is a device used to detect small amounts of radioactive contamination. The device has a gas filled chamber, which when it comes in contact with radiation becomes ionized and creates some voltage between a cathode and an anode - lots of beeping and clicking occurs. There are two ways that questions about this device are classically asked; (1) by just showing you a picture of one and saying "what is this", (2) by testing the concept of **"dead time."** *What is this "dead time" ?* Although the device is very sensitive, it is also vulnerable to being overloaded by a large dose of radiation. Then you have **"dead time"** as the ionization must dissipate before it can respond again. **The maximum dose it can handle is about 100mR/h.** So if the device clicks once and stops it might be because of "dead time" (it might also be "dead time" for you).
- * **Ion Chamber** - This is what is **used when higher doses are expected.** You don't have the dead time problem, for reasons that don't matter. Ion chambers can be used to detect exposure rates from **0.1 to 100R/h** (*note the unit change*). The dose calibrator used in most nuclear medicine departments is actually an ionizing chamber.



Geiger Muller
- With pancake probe
- Detects ionizing radiation
(*alpha, beta, and gamma*)



Ion Chamber
- For measuring dose rate
- Used with higher rates

Geiger - Muller Counter	Ionizing Chamber
Very Sensitive	Lower Sensitivity
Great for Low-Level Radioactive Survey	Stable across a wide voltage range - Excellent for accurate estimates (or exposure).
Terrible for Very High Radiation Fields ("Dead Time")	

- **Sodium Iodine Well Counter** - This is basically a small gamma camera, with just one PMT. There is a hole in the block of NaI crystal into which the sample is placed (so it's surrounding the sample). So this gives you great efficiency in detection, but has a problem with being overwhelmed. If the sample exceeds 5000 counts per second (a lot less than a micro curie), it will be under reported. It's good for in-vitro blood or urine samples. It's good for "wipe test" samples.



- * **Thyroid Probe** - This is a modified version of the NaI well counter. Its primary use is to calculate thyroid uptake values. The device has shielding, with a small opening that is pointed at the patient, at a precise distance. Dose is compared to a calibrated capsule of the same radionuclide.

Device	Trivia
Personal Dosimeters	<p><i>“Pocket Ionization Detector”</i> - uses a miniature ionization chamber. They give you real-time estimated dose, but must be charged and zero’d prior to use. These are not used anymore - which makes them high yield.</p> <p><i>“SolidState Dosimeter”</i> - Accumulated dose or rate can be read real time with LCD display.</p> <p><i>“Film Badge”</i> - Uses a thin metallic filter with a radiosensitive film. The degree of darkening (relative optic density) corresponds with dose. They can be damaged by temperature, humidity, etc...</p> <p><i>“Optically Stimulated Dosimeter”</i> - Replaced the film badge. Chips / Strips are placed under a filter.</p> <p><i>“Thermo-luminescentDosimeter”</i> - The ring badge. Should be worn under a glove, with the label facing the palm (target).</p>
Survey Meters	G-M and Ionization Detectors - discussed above
Well Counter	Basically a small gamma camera, with one PMT. Susceptible to “dead time” at counts over 5000 per second. Good for urine and blood samples. Good for “wipe test” samples.
Dose Calibration and Automated Dose Injection Systems	Used to measure radiopharmaceuticals.
Thyroid Uptake Probe	Compares counts from region over the thyroid to a calibrated capsule of the same radionuclide. The probe is a cylindrical scintillator detector attached to a PMT. A positioning guide keeps the distance constant
Intra-operative Probes	Used for lymphoscintigraphy

Q/A on the Dose Calibrator (Ionizing Chamber): Readout of activity is made in mCi. The range of most devices is 30 microCi to 2 Ci. Dose **should be within 5% of computed activity**, and this **should be checked daily**.

- * *Consistency* - should be within 5% of computed activity. Checked with reference sources (checked **Daily**)
- * *Linearity* - accurate readout for activities over the whole range of potentially encountered activities - checked with a large activity of Tc (around 200mCi) and decaying it down to less than the smallest activity you would measure for use. Or - the easy way - use a Calicheck© or Lineator© kit (which contains sheets of varied thickness of lead, simulating decay over time), (checked **quarterly**).
- * *Accuracy* - Standard measurements of radiotracers measured and compared to what the activity should be (performed **at installation of the device and annually**).
- * *Geometry* - Correction for different positioning and size (different volumes of liquids) of the sample (performed **at installation and any time you move the device**)

Regulations:

No bureaucracy is complete without lots of rules and regulations (and of course agencies to police such regulations). The Code of Federal Regulations (CFR) is where you will want to look for all your rules and regulations. Specifically part 19 (inspections), part 20 (radiation protection), and part 35 (human use of radioisotopes). The “NRC” is the governing body that has been charged with the task of enforcing all these various directives. It is possible for individual states to reach an agreement with the Federal Government to enforce these rules on their own. These are called “**Agreement States**”, and the main thing to know is that **they can be more strict, but not less strict than the national agency**.

Radiation Safety / Contamination

Accidents happen. Rules to know for answering questions about spills:

First Things First - is it a MAJOR spill or a MINOR spill ? (**HIGH YIELD SECTION**)

Major Spills

- * Activity level greater than **100 mCi of Tc-99m** is considered a major spill.
- * Activity level greater than **100 mCi of Tl-201** is considered a major spill.
- * Activity level greater than **10 mCi of In-111**, is considered to represent a major spill.
- * Activity level greater than **10 mCi of Ga-67**, is considered to represent a major spill.
- * Activity level greater than **1 mCi of I-131** is considered to constitute a major spill.

Radiation Safety Officer needs to be notified immediately when a major spill occurs, to direct the decontamination process.

Minor Spill = You Clean It Up

Major Spill = Don't Clean it Up, Call the Radiation Safety Officer

Minor Spills

- (1) **Protect the Patient:** If a spill occurs while a patient is in distress, address the patient first. Once he/she is stable then address the spill.
- (2) **Confine the Spill / Limit the Spread:** Secure the area and make sure people aren't tracking it all over the place.
- (3) **Clean Up the Spill:** Use gloves (wear shoes), and all other personal protective equipment including a radiation badge. If possible tongs or forceps are even better for grabbing stuff. Use a damp absorbent material to clean the spill (working from the outside to the center).
- (4) **Survey Cleanup Items:** Anything used in the clean up needs to be surveyed or presumed contaminated (held until they decay to safe levels - rule is 10 half lives).
- (5) **Survey Cleanup People:** People also need to be surveyed by the radiation safety officer (in a different area — to avoid count interference).

Major Spill - what do I do ???

1. Clear area.
2. Cover spill with absorbent paper. **Do NOT clean it up.**
3. Clearly indicate boundaries of spill area. Limit movement of contaminated persons
4. Shield source if possible
5. Notify the Radiation Safety Officer immediately
6. Decontaminate persons

What ifs:

- * *What if it gets on my clothes ? Take them off- they are contaminated. Clothes will be held by the RSO until activity has decayed to safe levels.*
- * *What if it gets on my skin ? Wash with soap and water (don't scrub so hard you break your skin).*

Obviously it is best if the Radiation Safety Officer (RSO) is involved and doing all that stuff. In the real world that is what will happen. But the ABR feels that on an intermediate level test you should be able to perform the tasks of doctor, tech, radiation safety officer, and janitor (while receiving the salary equivalent of the janitor).

Crazy Shit They Might Ask:

What if there is a Xenon leak?

- Without alarming the patient, instruct all individuals to leave room as quickly as possible. Close the door.
- Testable Trivia = The wipe test does NOT work on xenon contamination.

Regulations Affecting the General Public

Regulations demand the following:

- Annual Dose limit of 1 OOmrem to the public
- Not greater than 2mrem per hour - in an “unrestricted area”

Signs must be placed with the following slogans:

- *Radiation Area:* Any place you could get 0.005 rem (0.05mSv) in 1 hour at 30cm
- *High Radiation Area:* Any place you could get 0.1 rem (1mSv) in 1 hour at 30cm
- *Very High Radiation Area:* Any place you could get 500 rads (5 gray) in 1 hour at 1 meter

Occupational Exposure Dose Limits

The following are the rules:

- Total Body Dose per Year = 5 rem (50 mSv)
- Dose to the Ocular Lens per year = 2 rem (20mSv)
- Total equivalent organ dose (skin is also an organ) per year = 50 rem (500mSv)
- Total equivalent extremity dose per year = 50 rem (500mSv)
- Total Dose to Embryo/fetus over entire 9 months - 0.5rem (5mSv)

Recordable and Reportable Events

The dose you order should be the dose the patient gets (sorta). The limit is 20% from the dose via the NRC (and 10% in some agreement states). You want to be a Radiologist? You better get your vocab straight on this stuff. Bean counters take this stuff pretty seriously:

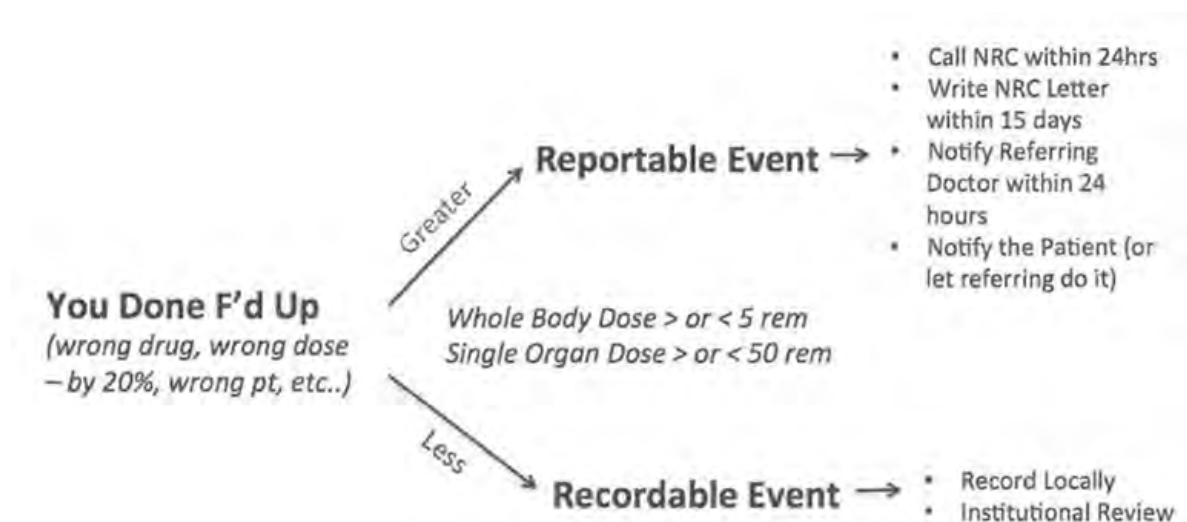
When does **a medical event** occur??? Well you have to meet two criteria:

- * (1) You have to “F” it up:
 - * Wrong drug, Wrong route, Wrong patient, or Wrong dose (more than 20%).
 - * OR Patient receives a dose to a part of the body other than the intended treatment site that exceeds by 50% or more the dose expected by proper administration and prescription
- * (2) You have to harm the patient
 - Defined as either: (a) whole body dose > 5 rem, or (b) single organ dose > 50 rem

Recordable Event	Diagnostic Medical Event
Whole Body Dose < 5 rem	Whole Body Dose > 5 rem
Single Organ Dose < 50 rem	Single Organ Dose > 50 rem

Medical Events require you to call the doctor who ordered it, patient, and NRC/State and explain what happen and if anything needs to be done about it.

Recordable events have to be documented (recorded) and kept for safe keeping - for 3 years.



Receiving, Storing, and Disposing of Radioactive Material

Within 3 hours of receipt (3 working hours) you (the tech) has to survey packages when they arrive. This process involves a GM counter test at the surface and 1 meter from the package, as well as wipes of all surfaces of the package (>6600 dpm/300 cm² is not allowed). Keep the package in a controlled area, like the hot lab (just in case A1 Qaeda invades your hospital). Contact shipper and the NRC if beyond allowable limits.

Package labels and allowable limits

White 1: No special handling, surface dose rate < 0.5 mrem/hr, 1 meter 0 mrem/hr

Yellow 2: Special handling required, surface dose rate < 50 mrem/hr, 1 meter < 1 mrem/hr

Yellow 3: Special handling required, surface dose rate < 200 mrem/hr, 1 meter < 10 mrem/hr

**these assholes couldn't pick another color? - instead they choose "yellow 3"*

"Transport Index" - "T.I." - This is the measured max dose at 1 meter. This is an actual dose rate measured at 1 meter (not an allowable dose rate), at the time of shipping.

- *Radioactive Label 1:* White 1 -
There is no T.I. because the rate at 1 meter will be so low.
- *Radioactive Label 2:* Yellow 2 -
The T.I. is < 1.0 mR per hour.
- *Radioactive Label 3:* Yellow 3 -
The T.I. is > 1.0 mR per hour.



"Common Carriers" - A truck that carries regular packages and radioactive material the T.I. should not exceed 10 mR / per hour. The surface rate should not be more than 200mRem.

"Multiple Packages" - Those shipped together; the sum should NOT exceed 50 mR.

— (switching gears gracefully) —

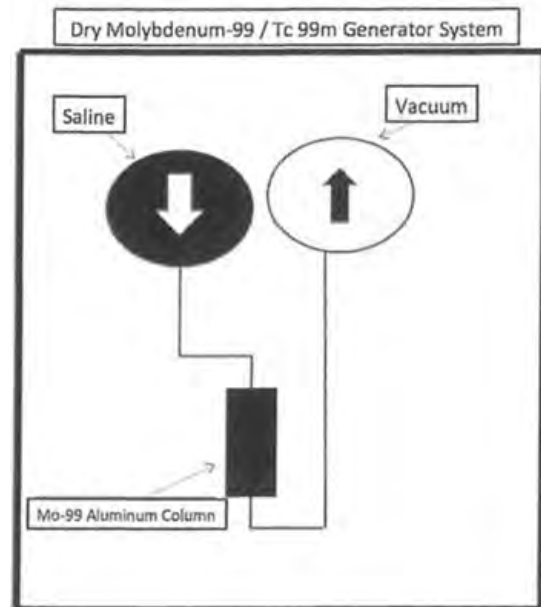
Tc-99

I now want to transition into discussing Tc-99. It's the workhorse of nuclear medicine, and its production / purity is likely high yield for multiple choice.

First let's talk about how Tc is made.

Step 1 "Mo / ^{99m}Tc Generation"

Because Molybdenum's half life is longer than Tc ($Mo = 67 \text{ hours}$, $Tc = 6 \text{ hours}$), it can be made and shipped in a Tc generator. Mo adheres to the aluminum column tightly. As it decays it does not stick as tightly and can be washed off with saline across the column. When it comes out the Tc is stuck to Na ($Na^{99m}TcO_4$). The piece of trivia to know, is that it's in a +7 valence state, and must be reduced to be used. This is accomplished with stannous ions.



Step 2: Radionuclide Purity

You want to make sure you just have Tc, and you didn't wash any Mo off as well. Mo that is in the sample is called "*break through*." The basis for the test is to look for the different photo peaks of the radionuclides. The sample is placed behind a lead shield. Mo is assayed for first. The basis of the test is that the high energy photons of Mo (like 740 keV) will NOT be attenuated by the shield, but the 140 keV Tc photons will be.

* NRC allows **no more than 0.15 micro Ci of Mo per 1 milli Ci of Tc, at the time of administration.**

o Few pitfalls here: (1) note the unit change from micro to milli, (2) note the "time of administration"

* *If ratio is less than 0.038 at the time of elution, the material will be suitable for injection for a least 12 hours.*

Step 3: Chemical Purity

Remember that the column is made of Aluminum Oxide, and that can wash off, clump up with the Tc and show up as liver activity, or cause sulfur colloid aggregation and show up in the lungs on liver spleen scan. **The test for purity is performed with pH paper. The allowed amount is < 10 microgram Al per 1 ml**

Aluminum Contamination Can Be Shown Two Ways:

- (1) Liver Spleen Scan + Lung = Aluminum Contamination
- (2) Tc scan + Liver Activity = Aluminum Contamination

Step 4: Radiochemical Purity

As mentioned above Tc comes out of the generator as $\text{Na}^{99\text{m}}\text{TcO}_4$. So, to use it for anything useful it needs to be reduced (accomplished by adding it to SnCh). Thin layer chromatography is used to assess for purity.

Limits for Free Tc

- * 95% $\text{Na}^{99\text{m}}\text{TcO}_4$
- * 92% > for $^{99\text{m}}\text{Tc}$ sulfur colloid (MAA)
- * 91% for all other Tc radiopharmaceuticals

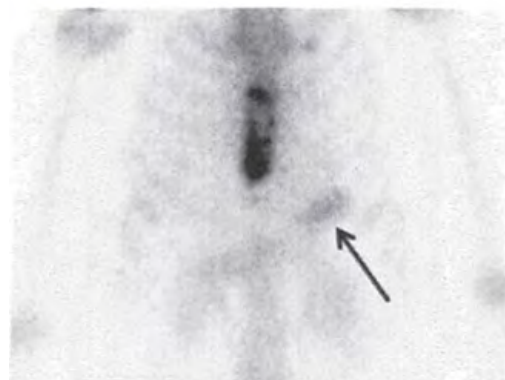
Some Sneaky Sinister Stuff:

They can ask questions in a variety of ways about this stuff but here are a couple of key points:

- * When you check the radiochemical Purity of $^{99\text{m}}\text{Tc}$ -MAA you are checking for free pertechnetate
- * Testing for Chemical Purity is NOT mandatory in NRC States
- * The ^{99}Mo and $^{99\text{m}}\text{Tc}$ ratio must be known at **the time of ADMINISTRATION**, not elution
- * When you are checking for Radionuclide Purity (breakthrough Mo), you have **to assay for Mo FIRST**, before the Tc (to prevent issues with residual charge).
- * Technically $^{99\text{m}}\text{Tc}$ made in a generator is not considered carrier free because of the presence of ^{99}Tc which is technically a radionuclide (but essentially stable since it's half life is 200,000 years).

Free Tc

Free Tc can occur from a lack of stannous ions (reducing agents), or accidental air injection into the vial or syringe (which oxidizes). The way it's shown on images is Tc scan with **gastric uptake, salivary[^] glands, and thyroid.**



Free Tc: - Gastric Uptake on bone scan
*incidental note of sternal met from breast ca

Vocab	What is it?	Tested?	Limit?
Radionuclide Purity	How much Mo in the Tc ?	Tested in a dose calibrator with lead shields;	0.15 microcuries of Mo per 1 millicurie of Tc
Chemical Purity	How much Al in the Tc ?	Tested with pH paper	< 10 micrograms Al per 1ml
Radiochemical Purity	How much Free Tc?	Tested with Thin Layer Chromatography	<ul style="list-style-type: none"> * 95% $Na^{99m}TcO_4$ * 92% for ^{99m}Tc sulfur colloid (MAA) * 91% for all other Tc radiopharmaceuticals

Equilibria

Equilibrium: Concentration of parent and daughter isotopes are equal.

Transient Equilibrium: This type of equilibrium occurs when the half life of the daughter is shorter than the parent (but not by a lot). The **classic example is the MoIy-99 generator making Tc-99**. A transient equilibrium occurs after 4 half lives (usually).

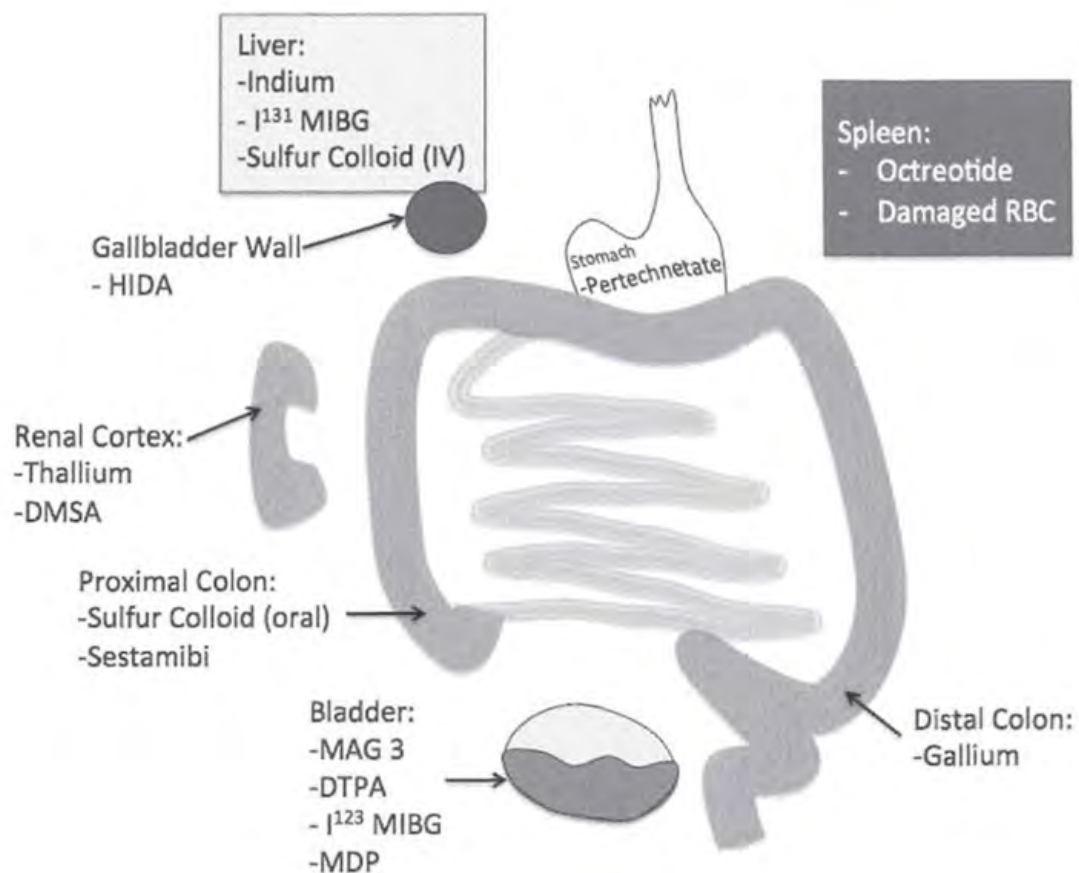
Secular Equilibrium: This type of equilibrium occurs when the half life of the daughter is way way way shorter than the parent.

Critical Organ

I want to define two frequently used terms:

- * “Critical Organ” - An organ that limits the dose of the radiopharmaceutical due to the increased susceptibility of the critical organ for cancer. This may or may not be the Target Organ.”
- * “Target Organ” This is the organ you want the tracer to accumulate in. It’s your organ of interest.

A few **generalizations on Critical Organs:** If you are trying to figure it out, it’s the organ that the tracer is going to spend the most time in. For example Gallium ends up in the bowel. Tc RBC scans are going to end up passing through the heart a lot, unless they are heat treated then the spleen eats them. Tc-MAG3s and DTPA is going to be the bladder, but **DMSA (which sticks to the kidney) is going to be the kidney.** For anything that uses free Tc (meckels scan), it’s going to be the thyroid. MIBG scans are going to hit the bladder (or thyroid if it’s not blocked).



SPECT

SPECT provides a 3D look at isotopes in the body. SPECT uses a mounted parallel hole collimator that rotates around the patient. Each projection takes like 30 seconds and the total scan is around 15 mins. The matrix size is usually around 128 x 128 (for things that are not cardiac). The scan uses iterative reconstruction to make a picture. Because the data is isotropic volume, reconstruction in 3 planes is possible. Sensitivity is depth dependent, with radiation from different tissue origins attenuated to different degrees. Special collimators with longer holes are used to help improve the collection of photons.

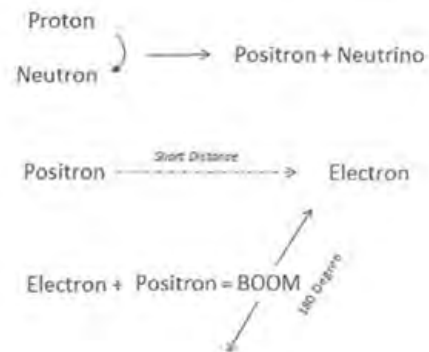
Testable Point: The big advantage SPECT has over planar is improved contrast from overlapping structures.

Testable Point: SPECT is depth dependent (PET is not).

Tuning Fork Artifact: Cardiac SPECT uses a 180 orbit. When a point source is imaged, it should look like a point source. If there is an error with the center of rotation (misregistration error) then it will look like a tuning fork (two lines in one direction and one line in the other). The same appearance can be seen with motion.

PET

As mentioned previously beta plus decay results in a proton being “transformed” into a neutron. This transformation requires ***1.02MeV for this to occur***. A positron is then emitted which travels a short distance before colliding into a real electron, whereupon the electron and positron destroy one another. The mutual destruction emits **two 511 keV photons which come out 180 degrees apart**.



Detector System:

You can't use the normal NaI crystals for a 511 keV energy photon, they aren't strong enough. Instead Lutetium Oxyorthosilicate (LSO) and Gadolinium oxyorthosilicate (GSO) are used.

Coincidence Timing:

The positrons shoot out in opposite directions (180 degrees) and collide on the detector at the same time. If they don't land at the same time (within nanoseconds) they are not counted. This is called “***coincidence timing***” This provides a type of virtual electronic collimation - giving PET an advantage over SPECT (which uses parallel hole collimators).

Spatial Resolution:

Spatial resolution in PET depends on (1) detector resolution and (2) positron range / angulation. Range is determined by photon energy and is characteristic of what they came from (F^{18} is 2.8mm, C^{11} is 3.8mm). Scatter and random coincidences cause degradation of image quality.

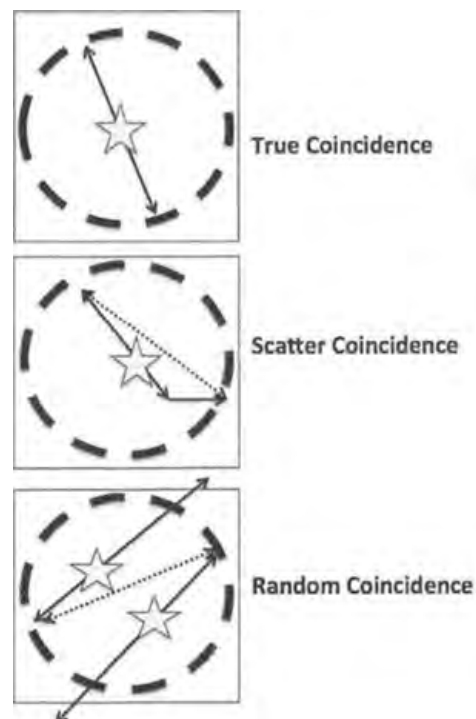
Sensitivity: PET CT has way better (10x - 20x) sensitivity compared to SPECT. This is because PET uses multiple pairs of detectors.

Scatter - There are three types of events detected by a PET scanner: (1) True, (2) Scattered, and (3) Random.

True Coincidence - Two 511 photons resulting from an annihilation reaction are detected in the same coincident window.

Scatter Coincidence - One of the photons has a Compton interaction and is deflected, but still hits the detector within the coincident time window (just not in the calculated location).

Random Coincidence: Two photons, from different annihilation reactions just so happen to land within the same coincident window - creating the false calculation that they occurred from the same event.



Rejecting Scatter:

As mentioned above, you can reject scatter based on the incident time on the detector.

Another way you can also reject scatter is based on photon energy. In the perfect world, both photons should measure 511. If the photon undergoes scatter it will lose energy, so you can exclude scatter by saying you will only keep a photon at 510 keV or above. However, if you exclude things that are just barely off of the 511 mark you will exclude a ton of true events as well. So, it turns into a balance of sensitivity and specificity. A tighter window gives you a more specific exam, but you are going to lose a lot of normal counts (and decrease your sensitivity). The opposite is obviously true as well.

2D vs 3D PET:

The modern scanners are “3D” and use the incident time to exclude scatter. Older scanners are “2D” and use septa to deal with scatter. This is the main difference, and only thing I can imagine could be tested.

Time of Flight PET

By measuring the difference in arrival times between the two photons from an annihilation event, a Time of Flight image can be created. TOF *can enhance spatial resolution and image contrast.*

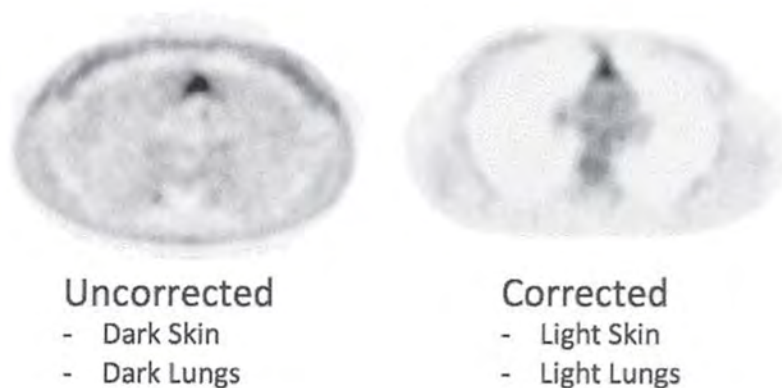
Attenuation Correction:

The CT part of PET-CT is performed for two main reasons: (1) so that you can see where things are and (2) for attenuation correction.

Attenuation correction is a correction for the different levels of attenuation a photon might undergo as it tries to get out of the body to the detector. Think about traveling through bone vs traveling through lung. The classic trick is the pacemaker (metal) making something appear really really hot - when attenuation is corrected for. This is why you always look at the uncorrected data, when reading a PET.

Which one is the uncorrected?

A sneaky trick is to ask you to distinguish between corrected and uncorrected PET. The secret is to look at (1) the skin - which will be hot on uncorrected, and (2) the lungs - which will also be hot on uncorrected.



Testable Point: Attenuation in PET is depth independent.

Calculation of Standard Uptake Value (SUV):

SUVs are not like H.U. (calibrated to water). There are multiple factors which go into the calculation of the SUV, and essentially make it highly variable from institution to institution and even patient to patient.

The calculation is this:

$$\text{SUV} = \frac{\text{Tissue Radioactivity Concentration at Time Point 1} \times \text{Patient Weight}}{\text{injected dose activity}}$$

Fat vs Skinny: Fat has a low FDG uptake. So, it's actually more accurate to use the patients lean body mass instead of weight (this is why the value is different from patient to patient).

SUV values in fat people are overestimated (there is more sugar around for the tumor) - this can be corrected for by using lean body mass.

Timing: The longer you wait between FDG administration and imaging the more FDG uptake you get. At first the uptake of FDG in the tumor is rapid (rapid in first 2 hours), then it goes up slower. In other words, if you did "delays" you would get higher FDG values. Another point is that if scan (1) is done after an hour, and scan (2) is done after 2 hours, then scan 2 will falsely appear to have increased SUV values. Basically, you need to do the scan at the same time interval after FDG administration if you want to compare apples to apples.

Glucose Levels: This is one of those competitive kinetics things. The more non-labeled glucose is floating around, the less FDG the tumor can drink. **High glucose = Lower SUV.**

Size Matters: There is a size threshold for PET (usually 1cm). Anything smaller than 1cm, will be subjected to partial volume effects, and give a false low SUV. **Smaller than 1cm = Lower SUV.**

Dose Extravasation: FDG is given IV (most things are - except xenon you breath and Iodine you eat). If you put all your FDG in the soft tissues of the arm, you have less circulating and get lower SUV. **Dose Extravasation = Lower SUV.**

Reconstruction Type: Iterative reconstruction can mess with the SUV values. The more iterations the higher the SUV.

Attenuation Correction: Attenuation correction with CT, also makes an adjustment for SUV values (the positrons have to go through denser stuff too). The method used for the computer to calculate attenuation correction also varies and can make comparing SUVs difficult.

Truncation Artifact:

This has to do with differences in FOV from PET and CT. The classic example is a giant monster fat person who is so fat that they have several feet of blubber outside the FOV (and therefore not providing data for attenuation correction). Bottom line = Giant Fat People can have artificially lower SUV.... Although the presence of fat may elevate SUV (as described above). So, maybe it will cancel out and be normal. **Just say Truncation Artifact = Falsely Lower SUV.**

Pre FDG PET prep:

Diet - Fasting for 4 hours prior to the test is the typical recommendation. If they just ate, they will spike their insulin and drive all the FDG into their muscles. Minimizing cardiac activity (you might want to do if you have a thoracic cancer) can be done with a 12 hour fast, and a low carb/ high protein & fat diet for 24 hours.

Hydration - Oral hydration, and frequent voiding decreases the dose to the bladder and improves urinary visualization.

Muscle Uptake - There are a couple of causes for diffuse muscle uptake; exercise (most places discourage this for 24-48 hours prior to the exam), eating or insulin use. More focal uptake (classic forearms from stress gripping) can also be seen.

Insulin - If you have to have insulin, long acting insulins are better to avoid diffuse muscle uptake. Metformin is ok - it won't mess with the muscles.

Brown Fat - Excessive brown fat uptake can be distracting. You can decrease it by (1) making the room warm, (2) giving drugs - with the big two being propranolol and diazepam (valium).

PET QA

Just like there is QA on all the other pieces of equipment in nuclear medicine, PET also has a whole bunch of QA. I've chosen two QA tests which I think are the most high yield. I believe they are high yield because their names sound similar and that means they'd make good distractors for each other on multiple choice.

Normalization Scan - This corrects for discrepancies in the thousands of detector elements. You scan a calibrated position source placed *in the FOV*. The scan serves to "normalize" the detection lines. This should be done once a month ("N-M" Normal Month).

Blank Scan - This is done to help keep the attenuation correction data accurate. This is done with *nothing in the FOV*. You simply use the systems transmission radiation source. I think about this as "zeroing" the scanner, or setting it as a "blank slate." It's done daily (you want to start each day with a blank slate).

High Yield Summary Chart

Tracer	Analog	Energy	Physical Half Life
Tc - 99m		“Low” - 140	6 hours
Iodine - 123	Iodine	“Low”-159	13 hours
Xenon - 133		“Low”-81	125 hours (<i>biologic t_{1/2} 30 seconds</i>)
Thallium - 201	Potassium	“Low”-135 (2%), 167 (8%), <i>use 71 ²⁰¹ Hg daughter x-rays</i>	73 hours
Indium - 111		“Medium” - 173 (89%), 247 (94%)	67 hours
Gallium - 67	Iron	Multiple; 93 (40%), 84 (20%), 300 (20%), 393 (5%)	78 hours
Iodine -131	Iodine	“High” - 365	8 days
Fluorine -18	Sugar	“High”-511	110 mins

Treatment Radionuclides Half Life	
Strontium 89	50.5 DAYS (14 days in bone)
Samarium 153	46 Hours
Yttrium 90	64 Hours

“Hell, I can get you a toe by 3 o ’clock this afternoon.....with nail polish ”

Section 9: MRI

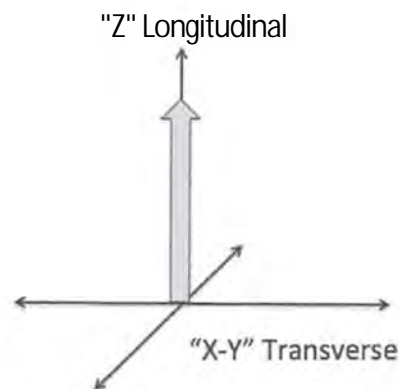
Although you can use other types of atoms, for the most part Hydrogen atoms (the proton) are what is being manipulated with MRI. Your body is full of water and fat, and therefore full of lots of hydrogen atoms (protons) that spins can be manipulated. Some people think about these protons as little tiny magnets.

How does MRI work?

The very basic answer is that you have a magnetic field - that is always on. Protons are normally randomly aligned, about half up and half down. When you stick someone in the magnet slightly more than half of them will align with the direction of the magnetic field. You can then slap them down with an RF pulse, and then watch how fast they bounce back up. The difference between how fast different protons bounce up is the “contrast” used to tell things apart - and make a pretty picture.

What is this “coordinate system” ?

Real quick basic point, is that when you see an arrow pointing up or down or sideways - this does not represent a single proton, but instead the addition of all the protons as a vector. Protons aligned in the direction of the external magnet field are called “longitudinal magnetization.” This is the “default setting” when someone is in the magnet. If they are aligned perpendicular to the Z axis, then they are considered “transverse magnetization.”



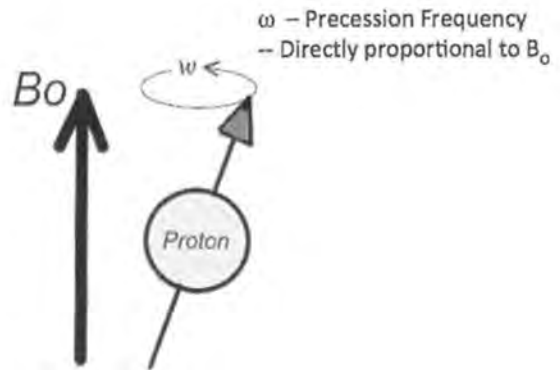
What is this “RF Pulse” ?

The RF pulse is a radio wave used to “knock the protons down.” To do this it has to push them. Pushing protons that are spinning in a magnet is a lot like trying to push your 5 year old on a swing. You have to wait until he’s swinging back at you and you push him as he is moving forward. In other words, you have to time it just right. Another way to think about this is if you wanted to hand someone something but they were running. You would need to run at the same speed as them to be able to hand it to them.

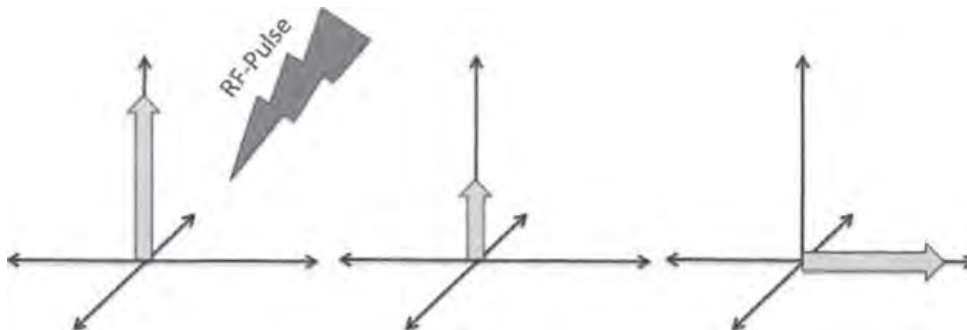
So how do you know how “fast to run” , or “when to push” ? There is this thing called the **Larmor Equation**.

$$\omega = \gamma B_0$$

- ω is the precession frequency
- B_0 is the external magnetic field strength given in Tesla.
- γ is a proportionality constant called the gyromagnetic ratio



The Larmor equation describes the precession frequency of a nuclear magnetic moment and resonant frequency of a nucleus, and relates these aspects to the magnetic field strength. It basically says the precession frequency gets higher as the field strength increases.



The RF pulse actually does two things: (1) it decreases the longitudinal magnetization, and (2) it causes the protons to synch up and precess in-phase (which establishes a transverse magnetization).

When can you measure signal?

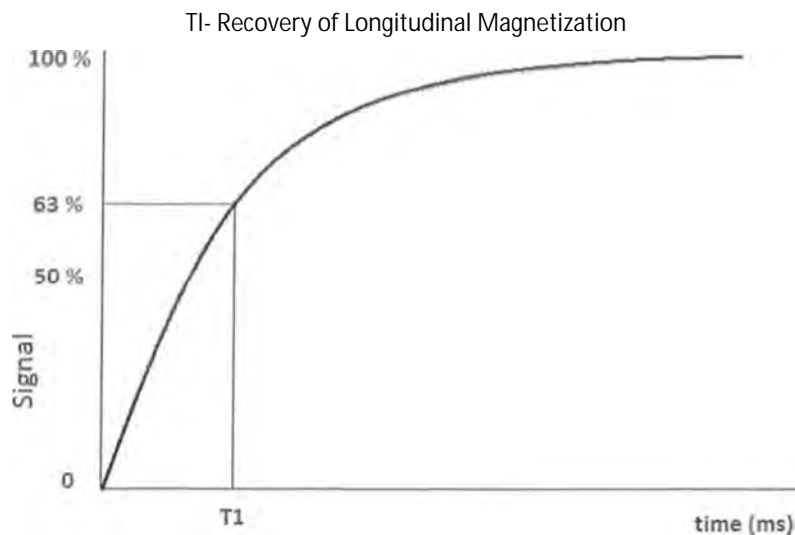
Signal can only be measured when it is **NOT in the longitudinal direction**. If you are running next to someone at the same speed, you can’t really measure how fast they are going (it’s like an Einstein relativity thing). But, if they turn and run away from you, then you can measure that. It’s sorta like that.

What is this T1?

After you knock the protons down with an RF pulse, they will grow back up to normal size (their spin magnitude will re-orient in the direction of B_0). The time it takes for this to happen is different in different tissues “called longitudinal relaxation”.

Some people call this “spin-lattice relaxation” because energy from the RF pulse is handed over to the surrounding lattice. The “1” of T1 resembles a thermometer - which is useful in remembering that T1 relaxation involves the exchange of thermal energy.

Plotting the time vs longitudinal magnetization creates the T1 curve.



The return to longitudinal relaxation follows an exponential curve approaching 100% over time. T1 is defined as the time at which longitudinal magnetization is **63% of its final value**. Each tissue has a different T1 and the **greater the field strength the longer the T1** (because net magnetization is greater in a larger field).

Things with short T1 signals are bright. Hence the phrase “intrinsic T1 shortening” is something that is bright on T1. Things that have long T1s are dark.

Is T1 different in a stronger magnet? A stronger magnet makes T1 longer. Protons in this stronger magnetic field have more energy (they precess faster), and therefore it takes longer to hand that over to the lattice.

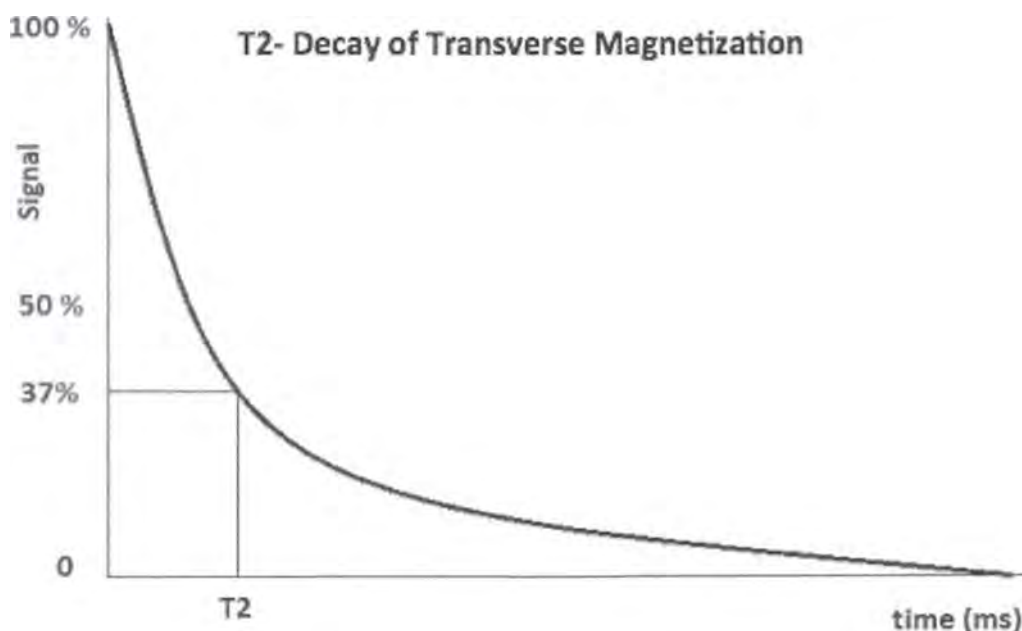
What is this T2?

Remember I said the RF pulse caused the protons to synch up and precess in phase (which establishes a transverse magnetization). As time progresses the protons will slowly fall out of synch and start doing their own thing again. This is the T2 Transverse relaxation.

They maintain net magnetization though because that is governed by B_0 .

Phase is effected by RF pulses.

Plotting the time vs transverse magnetization creates the T2 curve - which resembles the downward portion of a ski slope. I like this analogy because it helps me remember that T2 is shorter than T1 (takes less time to go down a hill than up it).



Transverse magnetization decay is also described by an exponential curve with T2 representing the time at which 37% of the original transverse magnetization value has decayed. Another term often used to describe T2 is “spin-spin relaxation.”

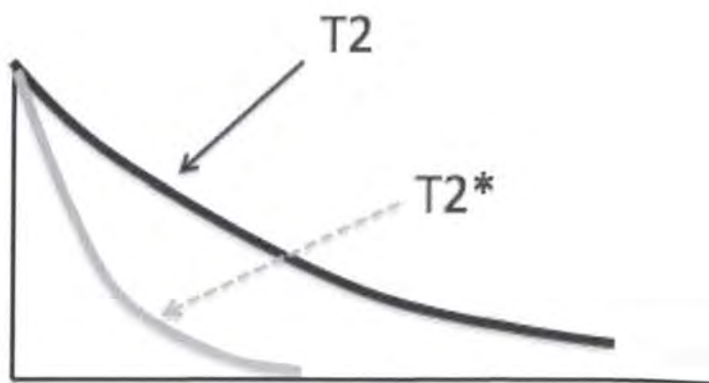
What causes protons to lose their transverse sync (T2 relaxation) ?

There are two main reasons (1) inhomogeneities in the external field, and (2) inhomogeneities in the local magnetic field - within the actual tissues. Pure things (water) with less inhomogeneities take longer to decay their transverse magnetization, and are therefore bright (the opposite is true of impure liquids).

T2 vs T2* ?

The signal of T2 decays faster than various PhDs would predict based on tissue spin interactions alone. The reason for this is that math falsely assumes the main external field is absolutely homogeneous — it is not. This heterogeneous field creates additional interaction which further speeds decay. It is because of this that **T2* decay is ALWAYS faster than T2**.

- $T2^* = \text{Tissue Spin Interaction} + \text{Field Inhomogeneity}$; “random + fixed causes”
- $T2 = \text{Tissue Spin Interaction}$; “random causes only”

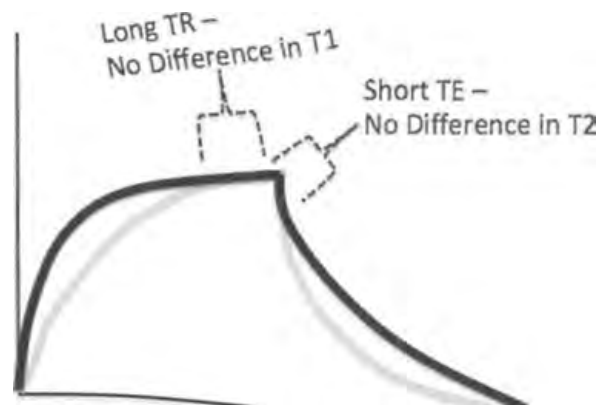


“Free Induction Decay” ? Without any magnetic gradient the received signal is called free induction decay.

Fixing $T2^*$ and making it $T2$? At 1/2 the time of TE a 180 pulse can be given to refocus (“turn around”) the signal which helps remove the fixed field Inhomogeneity.

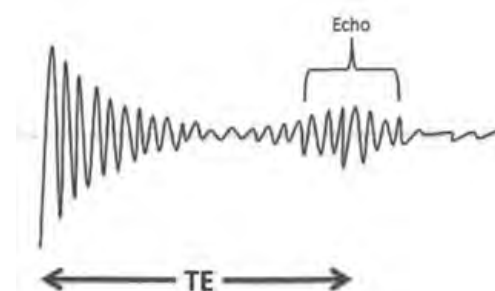
What is this Proton Density?

If you choose a long TR and a short TE the difference in magnetization recovery and in signal decay between fat and water is essentially nothing. In this situation the contrast is the result of differences in proton density. Tissues with more protons will have high signal. Tissues with fewer protons will have low signal.



What is this TR ? Repetition time (TR) - is the time between the initiation of two successive RF pulses

What is this TE ? The time between the middle of the 90° RF pulse and the peak of the detected echo.



T1	T2	Proton Density
Short TR	Long TR	Long TR
Short TE	Long TE	Short TE

The Longer the TE, the Greater the T2 Effects.

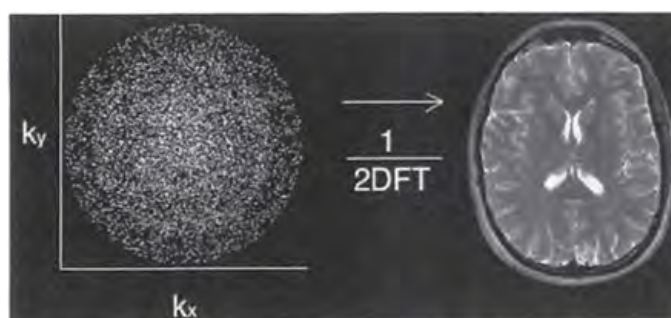
MRCP has a very long TE

	Short TR	Long TR	Short TE	Long TE
Spin Echo	250-700 ms	>2000 ms	10-25 ms	>60 ms
GRadient Echo	< 50 ms	>100 ms	1-5 ms	>10 ms

What is this k-Space and Image Matrix ?

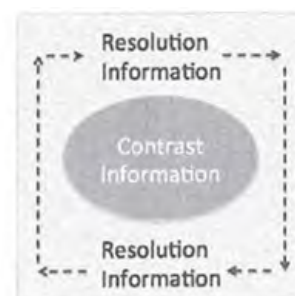
First let me introduce this thing called a “Fourier Transform.” A fourier transform is a mathematical technique for converting data from the time domain to data in the frequency domain.

K-space is a Fourier plane (like an x-y axis coordinate system) in which MR signal is stored. Turning K-space into an image requires an inverse two dimensional Fourier Transform (lots of math... which computers are good at).



Testable Point:

- The center of K-space is made from the information about gross form and tissue contrast.
- The periphery of K-Space is made up of information about spatial resolution.



Spatial Encoding

This vs That

Localization of signal requires three steps:

- (1) Select the desired slice
- (2) Encode spatial information along the rows
- (3) Encode spatial information along the columns

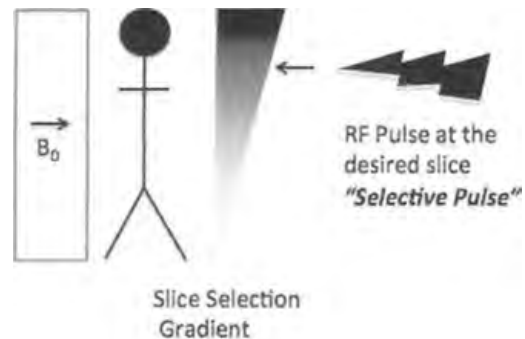
Gradient can be turned on or off.

Superconducting magnet is always on.

The localizing gradients have identical properties they are just applied at different times and different directions. Because you have three gradients in three planes you can localize anything in the body.

(1) Selecting the Desired Slice: The use of a slice selection gradient (SSG) is used to select the area of interest. This gradient is placed perpendicular to the desired slice plane. The slice selection gradient determines the view (axial, coronal, sagittal, even oblique).

Selective Pulse: On top of this gradient, an RF pulse is applied - at the same frequency as the protons in the slice plane you want to sample. That way only the protons in this plane will be affected. This pulse is called a selective pulse.



Dealing with the 180 degree refocusing pulse: In a spin echo sequence you apply a 180 RF pulse after your 90 degree pulse. Apparently this jacks up your nice field (especially along the edges). To deal with this, before and after the 180 degree RF you place two identical gradients (which cancel each other out) and correct for errors around the edges.

(2) Encoding Spatial Information in Vertical

Direction (Phase Encoding): This is the second step in the process. This gradient is applied causing protons in the same row perpendicular to the gradient to have the same phase. All protons at this point will have the same frequency.



Phase Encoding
Gradient

Phase encoding is much longer than frequency encoding - this is why it's done on the thinner portion. They don't want to lose time including the tip of the nose on phase encoding - this is why it's side to side in the head. The opposite is true in the abdomen, where it's front to back. Breast is an exception to this rule (I'll touch on that later).

Contribution to duration of study: The number of phase encoding steps contributes to the duration of a 2D imaging sequence.

$$\text{Duration} = TR \times N_{py} \times N_{ex}$$

TR = Repetition Time

N_{Py} = Number of phase encoding steps

N_{ex} = Number of excitations

(3) Encoding Spatial Information in the horizontal

direction (Frequency Encoding): This gradient is applied perpendicular to the phase-enc direction, which results in the modification of Larmor freqs over the duration of its application. The end result is column of protons which have identical frequencies.

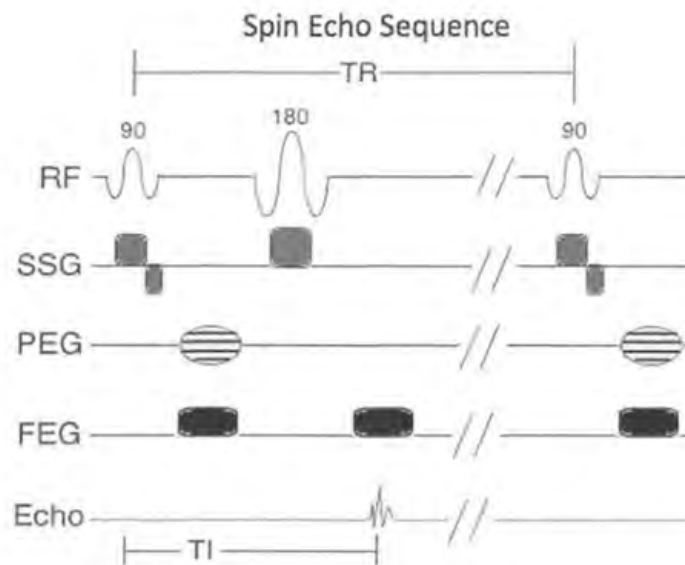
Testable Trivia: This is encoding gradient is applied at the same time as the readout.

For reasons that are complicated and confusing a “half lobe” frequency encoding gradient is applied in the opposite direction (dephasing) prior to read out to obtain the equivalent of a bipolar effect.



Frequency Encoding
Gradient

Lets walk through the Spin Echo Diagram to help solidify this process:



- (1) Slice Selection: This SSG is applied the same time as the 90 degree RF pulse (called a selective pulse).
- (2) Phase encoding and frequency encoding gradients are applied.
- (3) Next a 180 refocusing RF pulse is given. Before and after this 180 pulse two identical SS gradient lobes are applied to help correct undesired spin on the edge of the slice.
- (4) Lastly a second FEG is applied at the same time as the read out echo.

This process is repeated for as many phase encoding steps you do.

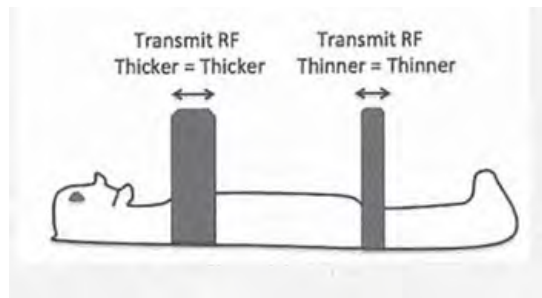
Slice Thickness:

Slice thickness is manipulated adjusting both the bandwidth of the selective pulse and the amplitude of the slice selection gradient.

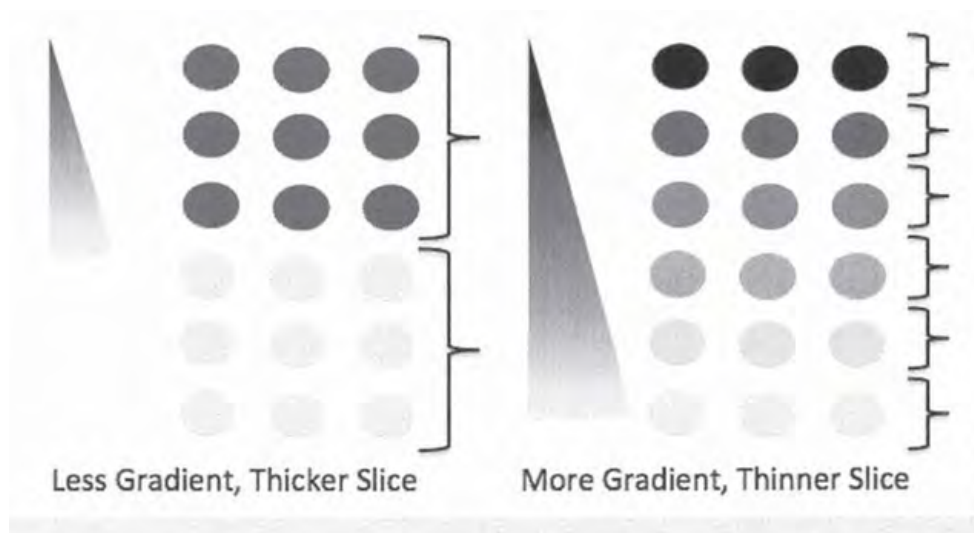
Slice thickness is governed by the following equation:

$$\text{Slice Thickness} = \frac{\text{Transmitted RF Bandwidth}}{\text{Slice Selection Gradient} \times \text{Some Random Constant}}$$

Transmit RF Bandwidth - Increasing the transmit RF bandwidth gives you a thicker slice.



Slice Selection Gradient - Decreasing the slice selection gradient gives you a thicker slice.



“Less is More” - Using a narrower RF pulse bandwidth will take longer to excite, resulting in a longer scan time. Thinner Slices = Longer Study.

3D Spatial Encoding:

Pulse sequences are either two dimensional or three dimensional. The three dimensional ones acquire volume from multiple sections in a single acquisition.

Spatial Resolution and Other Image Characteristics

Factors affecting spatial resolution: Spatial resolution is governed by the size of a voxel. The voxel size is determined by the matrix, field of view, and slice thickness.

$$\text{Voxel} = \text{Slice Thickness} \times \left\{ \frac{\text{FOV}_{\text{Phase}}}{\text{Matrix Size}_{\text{Phase}}} \times \frac{\text{FOV}_{\text{Read}}}{\text{Matrix Size}_{\text{Read}}} \right\}$$

Field of View: The smaller the field of view the better the spatial resolution. So, why not make the FOV super super small? Because you will get aliasing or wrap around artifacts from signal outside the FOV.

Matrix Size: Unfortunately, no one can be told what the matrix is, you have to see it for yourself. OK, fine the matrix size corresponds to the image width and height (in pixels). The larger the matrix the smaller the pixels, (pixel = FOV / Matrix)

Gradient: A gradient with higher amplitude (more intense) or one applied for a longer period of time results in better spatial resolution. Makes sense since the point of a gradient is to localize stuff.

Slice Thickness: For a thicker slice you can either increase the Transmit RF pulse (the slice selection pulse) or you can decrease the slice selection gradient. For a thinner slice you would do the opposite. The thinner the slice, the better the spatial resolution.

Factors affecting signal to noise ratio (SNR): Noise is random variation in the signal that leads to its degradation. In MRI the primary source of this noise is the patient's body. Overall the SNR depends on voxel size (bigger is better), field strength, and some random sequence parameters.

Voxel Size: Anything that makes your voxel bigger improves your SNR (the opposite of spatial resolution).

- Thicker slices = Higher SNR
 - Increased Transmit RF Pulse
 - Decreased Slice Selection Gradient
- Larger FOV = More SNR
- Smaller Matrix = More SNR

Field Strength: A stronger field gets you more signal. Greater Field Strength = Greater SNR.

RF Coils: Smaller surface coils improve your signal (increased SNR) compared to a coil within the scanner.

Number of Excitations per Slice (number of averages): The more excitation you perform the more signal you get (increased SNR). The main trade off is a prolonged imaging time. Longer TR, Shorter TE = Better SNR

Receiver Bandwidth: A fat bandwidth gives you a rapid sampling of data, a narrow bandwidth gives you slow sampling of data. Since noise is constant, the fatter band will pick up more noise. So, fat bandwidth = decrease SNR, narrow bandwidth = increased SNR.

Bigger Bandwidth isn't All Bad

As an aside, the larger receiver bandwidths allow for a higher frequency bandwidth per pixel which decreases mismatch artifacts like chemical shift or magnetic susceptibility.

Tradeoffs among spatial resolution, SNR, and acquisition time: In summary, each of these entities is affected by attempts to optimize the other.

For example, SNR can be increased by increasing field strength, but this increases tissue T1 times (and thus acquisition time). Spatial resolution can be increased by smaller voxel size, but this creates a noisier image.

Extremely High Yield Summary Table

Modification	Signal to Noise	Spatial Resolution	Duration of Exam
Thicker Slices	Increased	Decreased	No effect
Larger Field of View	Increased	Decreased	No effect
Larger Matrix	Decreased	Increased	Increased
Greater Field Strength	Increased	No effect	No effect
Greater Receiver Bandwidth	Decreased	No effect	Decreased
More Excitations per Slice	Increased	No effect	Increased
Utilizing Partial K Space Sampling	Decreased	No effect	Decreased

MRI Sequences

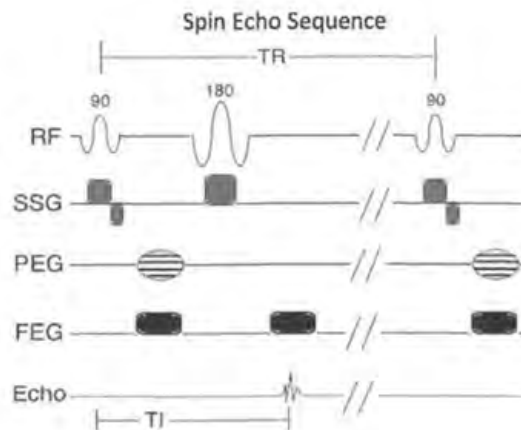
There are a bunch of sequences, but two general categories:

- Spin Echo - which uses a 180 degree rephasing RF pulse
- Gradient Echo

Spin Echo:

Spin echo is composed of a 90 RF pulse followed by an 180 rephasing pulse. This rephasing pulse is done at 1/2 the time of echo (TE). The sequence is repeated (TR) until k space is filled.

Why do you give this 180 pulse? In an ideal world the magnetic field is homogeneous, but we don't live in an ideal world. The pulse is **given to try and improve the heterogeneous nature of the field** and this is the reason spin echoes give us T2 and not T2*.



As discussed in the spatial encoding section, the slice selection gradient is applied with the 90 degree RF pulse. Then you have the Phase encoding and the frequency encoding gradients. The 180 degree pulse is flanked by self canceling slice selection gradients. Lastly the frequency encoding gradient fires again with the read out echo.

High Yield: $Duration = TR \times N_{py} \times N_{ex}$

TR = Repetition Time

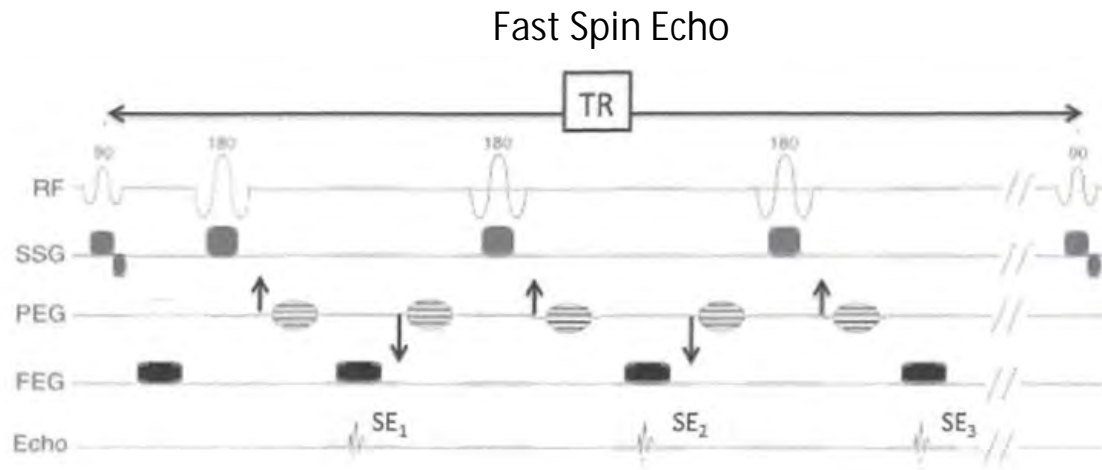
N_{py} = Number of phase encoding steps

N_{ex} = Number of excitations

Contrast: The contrast on spin echo sequences is determined by the TR (interval between 90 degree RFs), and the TE (interval between the 90 degree RF and the receipt of the echo). As mentioned above, a short TE and TR gives you T1 weighting, long TE and TR gives you T2 weighting, and long TR & short TE produce a proton density sequence.

High Yield Fact: Because a line in k space is filled at each TR, the TR contributes to the duration of the sequence. So, much so that long TR times are the reason a true spin echo is of historic significance.

Fast Spin Echo



The idea behind the FSE sequence is to reduce the TR, which I already mentioned is a major contribution to the duration of the study. This is done by applying multiple 180 RF pulses each resulting in an echo.

Echo Train Length - Number of echoes in the same TR.

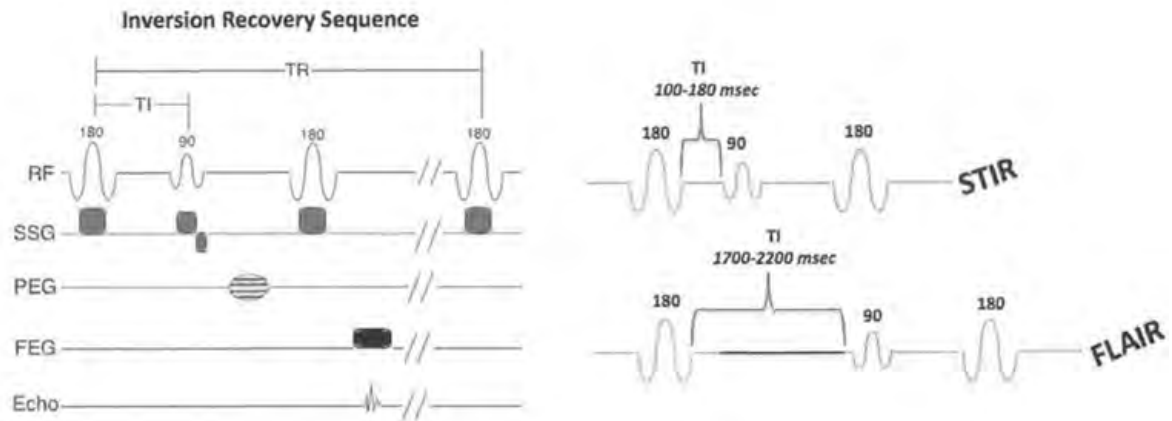
Fat Signal - There is a “normal” phenomenon called “*J coupling*” which occurs between the nuclei of lipid molecules, causing intrinsic shortening of T₂ signal. The fast repetition of 180 degree pulses mess up the J couples and cause the T₂ of fat to lengthen.

Testable Point: T₂ fat signal is longer with fast spin echo (interferes with J coupling).

Testable Vocab: With each progressive echo train the transverse signal gradually decreases. This is called “*T₂ Blurring*”

Testable Point: Acquisition time is approximately proportional to 1/ETL

Inversion Recovery

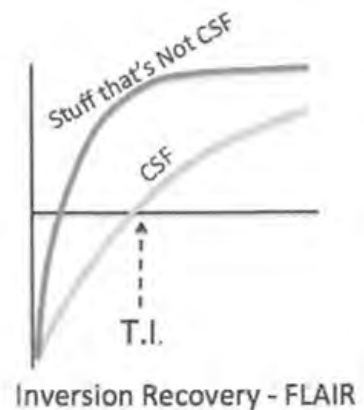


This time instead of starting with a 90 degree RF pulse, you start with a 180 degree “preparation” pulse. You wait for the relaxation of the thing you want to saturate (water, fat, myocardium) to hit its null point then you slam it with the 90 degree pulse. This way that particular tissue gives no signal.

“77” - The time between the 180 and 90 pulse.

“*STIR*” - Short T1 Inversion Recovery, employs a *short* TI (120- 160 ms) to suppress fat, which is based on the TI for fat. Often used on MSK imaging to suppress the fat within bone marrow to allow visualization of fluid (edema) within bone.

“*FLAIR*” - Fluid Attenuated Inversion Recovery, employ a TI designed to suppress water signal (approximately 2000 ms). Frequently used in Neuro-imaging to suppress CSF signal (based on the TI relaxation time of CSF) to allow visualization of edema or inflammation (i.e. in multiple sclerosis).



Testable Point: Relative to other fat suppression techniques - STIR is much less “susceptible” to magnetic susceptibility (metal) and field inhomogeneity

Testable Point: STIR can NOT be used with Gadolinium. Gd+ enhanced tissues have a similar TI as fat, so they may get totally nulled out.

GRE Sequences

Gradient echo sequences are different than spin echo sequences because they (1) use a flip angle less than 90 and (2) do NOT have a 180 pulse.

The advantage to this low flip angle is faster recovery, shorter TR/TE times, and a faster scan. The flip angle is used to determine how much transverse and how much longitudinal magnetization is going to be used.

Testable Point: Because there is no 180 pulse you are dealing with $T2^*$ (not $T2$). For the same reason GRE is more susceptible to susceptibility artifacts.

Testable Point: Gradient sequences have a lower Specific Absorption Rate (less heating).

If there is no 180 pulse, how do you create an echo? A bipolar readout gradient (basically a frequency encoding gradient) is used.

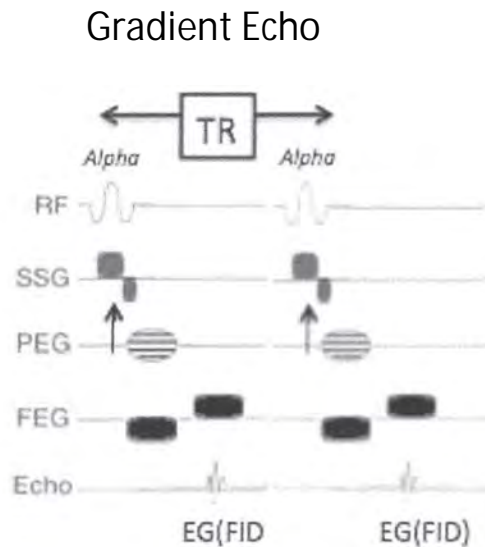
Vocab: An echo in gradient is called a “Field Echo”

If gradient is so fast and has a nice low SAR, why don't we use it for everything? The signal to noise ratio isn't that great. Plus, you get more susceptibility artifacts.

“Steady State” Because the TR is shortened in GRE you can get stuck with permanent residual transverse magnetization - where the magnetization never completely goes away. What is often the case is that the TR is shorter than the $T1$ and $T2$ of the tissues, so $T2^*$ dephasing dominates. The two main flavors in GRE imaging are classified depending on how this residual transverse magnetization is handled.

Spoiled (incoherent) GRE: Gradients and/or RF pulses are used to get rid of the transverse magnetization ($T2$) that is persisting in the steady state.

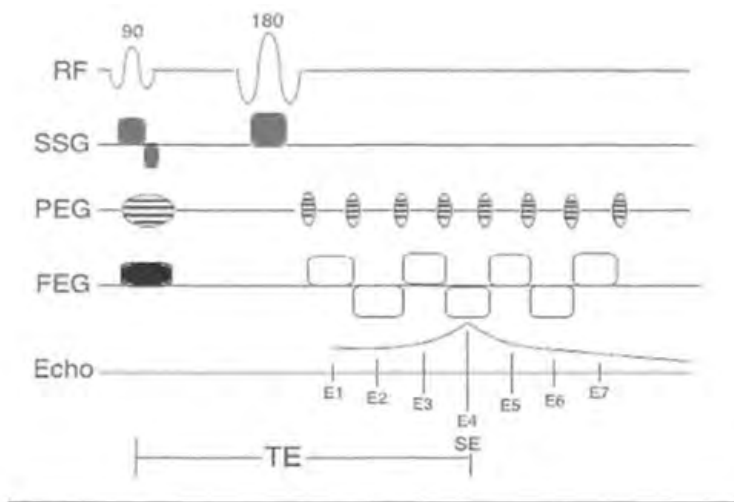
Refocused (coherent) GRE: The steady state is preserved by using a “Rewind gradient.” Therefore these sequences are $T2^*$ weighted. These refocused sequences can be partial or full. An example of the full refocus is the SSFP (steady-state free precession) which is used in cardiac imaging to provide a fast sequence with high signal to noise.



Spoiled (Incoherent) = Basically $T1$
Refocused (Coherent) = Basically $T2$

Echo-Planar Imaging (EPI) “The Noisy One”

Spin-Echo - Echo Planar Imaging



EPI can be done with spin echo (90 + 180) or gradient echo (90 + a bunch of gradients). EPI is MRI's fastest acquisition method, on the order of 100 ms/slice. One RF pulse is used to acquire data for an image (aka single shot).

The sequence works by turning the phase encoding and frequency encoding gradients on and off very rapidly - causing a very fast filling of k space.

Compared to a regular GRE: The sequence is more vulnerable to magnetic susceptibility, gives you better tissue contrast, and is faster.

Artifacts linked to EPI:

- Magnetic Susceptibility - can be improved with segmented sequences (instead of single shots)
- Ghosting - Gradient imperfections mess with spatial encoding
- Chemical Shift - A narrow readout bandwidth is used. As mentioned before, a fat readout bandwidth improves chemical shift (lets a bigger range in), a narrow one does the opposite.

Testable Trivia:

- Echo-planar is the technique of choice for Diffusion Weighted Imaging
- Echo-planar is highly vulnerable to magnetic susceptibility (even more than normal gradient sequences)

Diffusion

The base sequence is either a fast GRE or an echo planar. Generally speaking, diffusion weighted imaging works by using two very strong and symmetric MR gradients. The acquisition is repeated in each of the 3 dimensions in space with b-factors of 0 and b-factors of 1000. The signal differences are based on mobility and direction of water.

Scenario 1 (no net movement): The first gradient fires dephasing the spins. The molecules do not move. The second gradient fires rephasing the same molecules - giving you high signal.

Scenario 2 (net movement): The first gradient fires dephasing the spins. The molecules move out of the way. The second gradient fires missing the original protons - gives you low signal.

Vocab: The direction of diffusion is described as isotropic (movement in all spatial directions) and anisotropic (movement in a single direction).

“B-Factor” - The higher the “B-Factor” the greater the diffusion weighting. The “factors” of a B-factors include amplitude, duration, and spacing.

ADC (Apparent Diffusion Coefficient); - As I’m sure you know an ADC is needed to read diffusion correctly. The ADC map is calculated by obtaining (a) set of images without a diffusion gradient - poor mans T2, and (b) set of images with the diffusion gradient. A negative logarithm of the ratios is performed. Low signal is true restriction, high signal is T2 shine through.

Additional Sequences - Mentioned For Completeness:

Perfusion imaging

- Perfusion MRI quantitates cerebral micro-vascularization parameters. These parameters include regional blood flow and blood volume and mean transit time.

Functional MRI (fMRI)

- The basics underlying fMRI are increased blood flow to local vasculature that accompanies neural activity resulting in local reduction of deoxyhemoglobin. Deoxyhemoglobin acts as a contrast agent because it is paramagnetic (thus alters T2* MR signal).
- In fMRI procedures: First the patient performs a task, then Blood Oxygen Level Dependent (*BOLD*) *imaging* is done before and after the task. The pre and post task images are then subtracted and overlaid on a gray-scale brain image to localize the signal origin.

Angiography: 2D time of flight (TOF) MRA, 3D-TOF MRA, Phase contrast MRA

- **2D-TOFMRA:** Uses a gradient echo sequence where a saturation pulse is employed to null venous or arterial blood flow. Has a SMALL VOXEL SIZE.
- **3D-TOFMRA:** Is collected as a 3D volume, as opposed to slices, and allows for smaller voxels than 2D. 3D-TOF is well-suited for high flow arterial systems like the Circle of Willis. Benefits include a higher SNR than 2D, a shorter imaging time, more smooth vessel contours, and better saturation (which limits venous circulation).
- **Phase contrast MRA:** Uses bipolar gradients to create contrast from flow. High velocity encoding time (VENC) is needed for arterial imaging, and low VENC for veins and sinuses. Phase contrast MRA is a quantitative image and can measure mean blood flow velocity and direction.
- **In general:** TOF MRA is faster and less sensitive to signal loss from turbulent vessels than Phase Contrast MRA. Phase contrast MRA has advantages in increased background suppression and decreased sensitivity to intravoxel dephasing.

Fat Saturation Techniques

There are two broad categories.

- Inversion Sequences (STIR) that are based on the T1 of fat
- Those that exploit the resonance difference between fat and water protons.

STIR - As described above, this is a short inversion sequence (180, followed by a well timed 90). It does great with metal artifact. You can NOT use Gd with it.

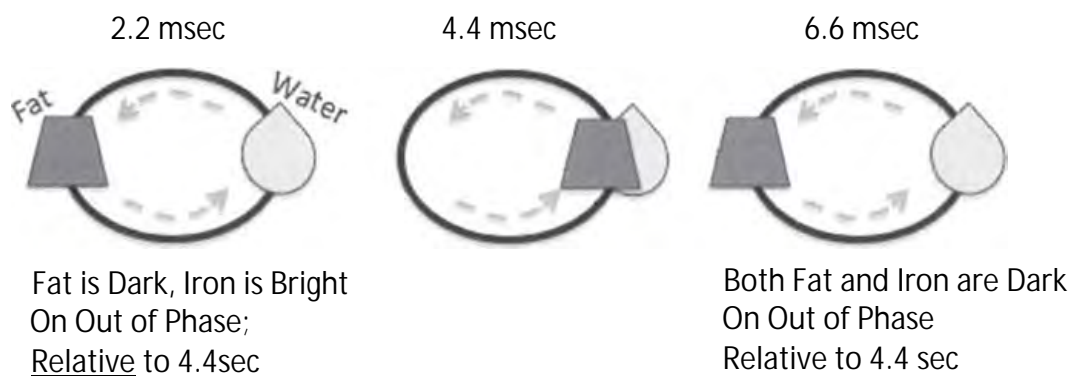
Selective Pulse: The protons in water and the protons in fat have different resonance frequencies. This difference can be exploited by delivering a selective RF excitation wave with a narrow bandwidth. The RF will only flip the fat protons, which can then have their magnetization destroyed by a “crusher” or “spoiler” gradient. This requires excellent field homogeneity, and can be used with most sequence types. This is the method used most commonly for contrast enhanced studies.

Selective Water Excitation: An alternative method is to use a combination of RF pulses (instead of just one) so you can flip the protons of water only. This technique also prefers a homogeneous field.

Fat Related Topics:

In Phase and Out of Phase: The chemical environments of fat and water are different for protons. This causes these protons to precess at different rates. A spoiled GRE is performed when the protons are spinning with each other (about 4.4 msec at 1.5T) and directly out of phase of each other (2.2 msec at 1.5 T). Microscopic fat will drop out on the out of phase.

High Yield Topic: Out of phase imaging at 2.2 seconds (on 1.5 T) **MUST** be done before in phase imaging at 4.4 seconds. If you compare the 6.6 second out of phase you will not be able to tell a fatty liver from an iron filled liver.



Chemical Shift Artifact: This will be mentioned again below, but I want to just touch on it briefly as it's related to fat. Again, because of the difference in proton environments between fat and water precession differences cause an artifact in the read out (frequency encoding) direction.

**Macroscopic Fat =
Chemical Shift**

**Microscopic Fat =
In and Out of Phase**

There are two subtypes.

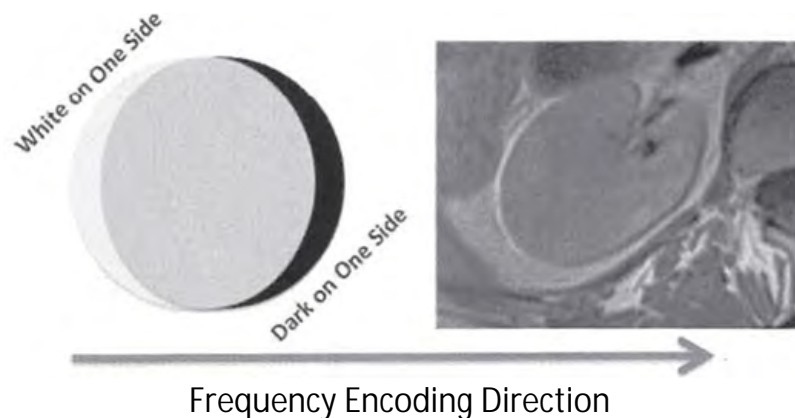
Type 1: This one manifestation is a bright rim on one side and a dark rim on the other. This can occur on either SE or GE sequences.

Testable Trivia: Chemical shift increases with field strength (it's not seen below 1 T)

Testable Trivia: Chemical shift decreases with increased gradient strength

Testable Trivia: Chemical shift decreases with a wider read out bandwidth

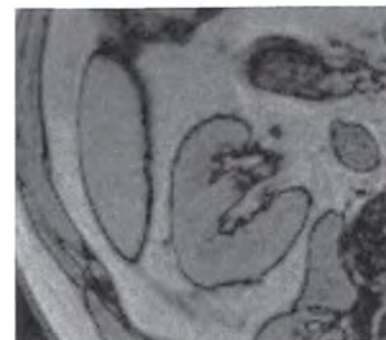
Type 1 Chemical Shift Artifact



Type 2: India Ink Artifact (Black Boundary) - This type of chemical shift artifact shows a black line in all directions of the fat-water interface. On Gradient Echo sequences, if a voxel has about 50% fat and 50% water the signals will cancel out - leaving this black line.

Testable Trivia: Using SE sequences will get rid of the India Ink, but NOT the Chemical Shift.

Type 2 Chemical Shift Artifact "India Ink"



MR Contrast (Gd⁺)

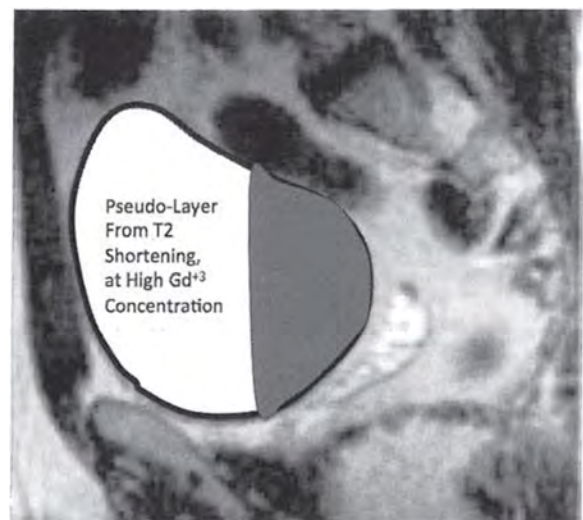
Broadly speaking MR1 contrast can be grouped into either “positive agents” which cause bright T1 signal (shortens T1), or “negative agents” which produce magnetic inhomogeneity from susceptibility leading the T2 shortening.

Gadolinium: This is a highly toxic metal that can cause all kinds of badness even in small doses - including liver necrosis. To solve this issue, Gd is complexed with a chelate. The toxicity, clearance routes, bio-distribution, and relaxation properties are all attributes to the chelating agent (DTPA).

How do Gd³⁺ cause a T1 shortening? Gd³⁺ has seven unpaired electrons, and the interaction of these ELECTRONS causes augmentation of the external magnetic field. The field of the electrons is very short so the contrast agent needs to be right next to the tissue to have an effect on it.

What about T2 effects ? At weak concentration the T2 effect is essentially nothing. However, at **high concentration** (classically seen as a “pseudolayer” the bladder) T2 effects dominate. MR1 Perfusion techniques use this T2 effect by using a concentrated bolus in the first intravascular passage - causing a drop in both T2* and T2 signal.

T1 Shortening = Bright
T2 Shortening = Dark



Extracellular Agents: These are the most clinically used agents that have a brief vascular phase, followed by an equilibrium in the extracellular space. *All of these agents are very hydrophilic.*

Blood Pool Agents: Keeping the agent in the blood can be done two ways (1) by using a large Gd ligand, or (2) by binding the Gd to a big sugar or protein molecule

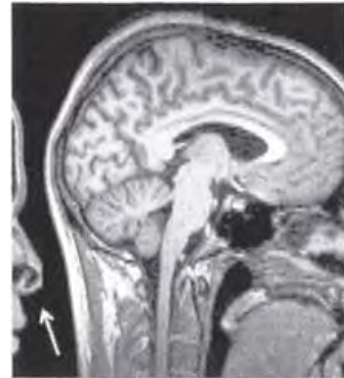
Pharmacokinetics:

- Gd cannot pass through a normal (intact) blood brain barrier
- Elimination is primarily renal
- Some agents (Gd-BOPTA and Gd-EOB-DTPA) have more excretion in the bile

MRI Artifacts

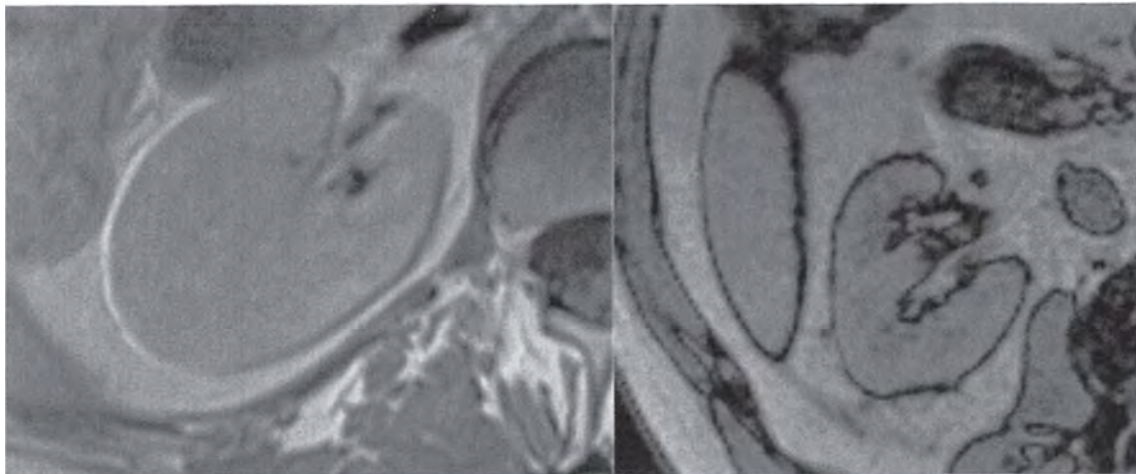
Image Process Artifacts: These include aliasing, chemical shift, truncation, and partial volume.

Aliasing: This error occurs when an area is under sampled. You get wrapping of anatomy from under sampled portions. This occurs in the phase encoding direction. You can correct for it by (1) increasing the field of view, or (2) changing the phase encoding direction. If you are dealing with aliasing in a 3D sequence then you can add slices or increase coverage to cover your field of view.



Aliasing- Nose Wrapped Around

Chemical Shift: As discussed above, the protons of different molecules precess at different frequencies. The difference between water and fat is around 220 Hz at 1.5 T (even worse at 3 T). This shift in the Larmor frequency is the so called “chemical shift.” The type 1 error occurs in the frequency encoding direction (opposite of most of artifacts which are P.E.).



Type 1

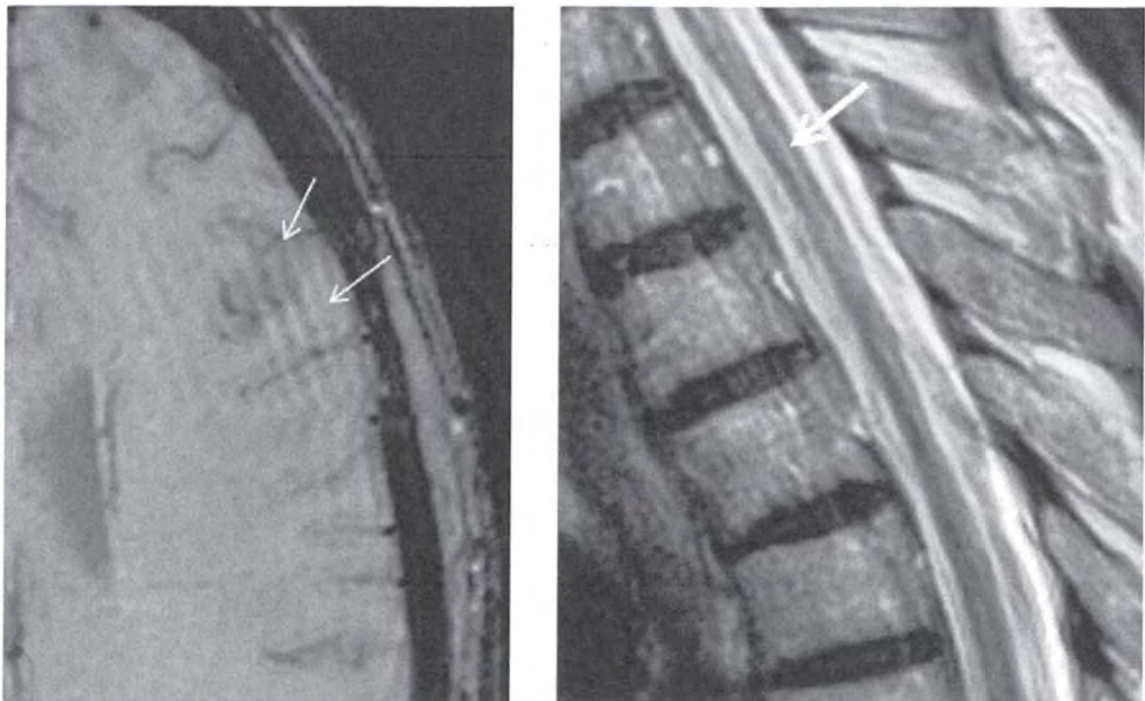
Type 2 - India Ink

Truncation / Gibbs: The transformation of K space through inverse fourier transform ideally (but never) resulting in a block of data. Ripples in this data - especially at abrupt intense tissue changes result in the appearance of lines. You classically see these at high contrast interfaces (skull -brain, Cord-CSF, and meniscus/fluid). **The CSF-Cord interface is the most classic - mimicking a syrinx.** If prompted I would say the cause is limited sampling of free induction decay. *It can be seen in both the frequency encoding and phase encoding directions* but is **more commonly seen in the phase encoding** because many times a phase encoding matrix that is smaller than the readout matrix is selected to reduce time.

Gamesmanship: The classic way to show this is sagittal view of the spine - looking like a syrinx.

Making it better: Short answer = **more matrix**. Long answer = Decreasing the bandwidth or decreasing pixel size (more PE steps, less FOV, more matrix).

Improvement penalty: Increased acquisition time and reduced per-pixel signal to noise.



Gibbs at the Skull -Brain Gibbs - Mimicking a Syrinx

Partial Volume: This is just like in CT. You have different signal intensities in different adjacent structures. If they overlap in a signal voxel - you get averaging. This can result in an intermediate signal from mixing of high and low intensities.

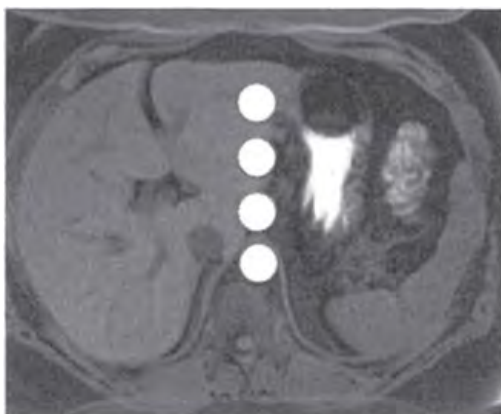
Making it better: Make your pixels smaller. More slices in the z-direction.

Patient Related Artifacts: These include Motion and Magic Angle

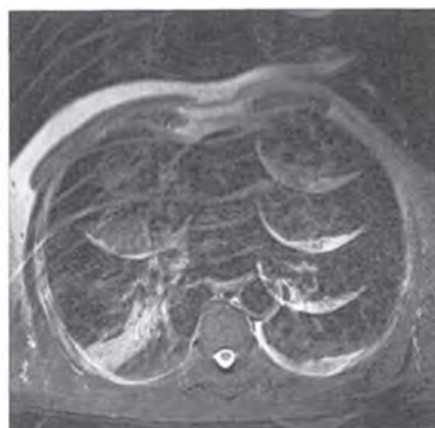
Motion: This can be seen with both voluntary and involuntary (cardiac, respiratory) movements. Motion creates difference between frequency encoding (which is fast) and phase encoding (which is slow). You will see ghosting or smearing - primarily in the phase encoding direction. You can also see the classic pulsation artifact from the aorta.

Making it Better:

- Perform Breath Holding / Having the Patient Hold Still - This is the best method
- Respiratory Gating (increases acquisition time)
- ROPE (Respiratory Ordered Phase Encoding) - Phase encoding steps are ordered with respiration
- Breathing Navigator - An echo from the diaphragm determines its position, then timing and acquisition are based off this.
- Apply a fat sat band across the abdomen
- Switch the phase encoding direction.



Pulsation Artifact – From the Aorta



Breathing Artifact

Flow: This is a type of motion artifact, related to blood flow. Blood flow causes ghosting in the phase encoding direction. GRE sequences are more susceptible to this than SE sequences.

SE Sequences - Flow looks **dark**. Moving blood that got hit with the 90 pulse, moves out of the way prior to getting the 180 pulse. Therefore, it doesn't have any signal.

GRE Sequences: In flowing blood looks **bright**.

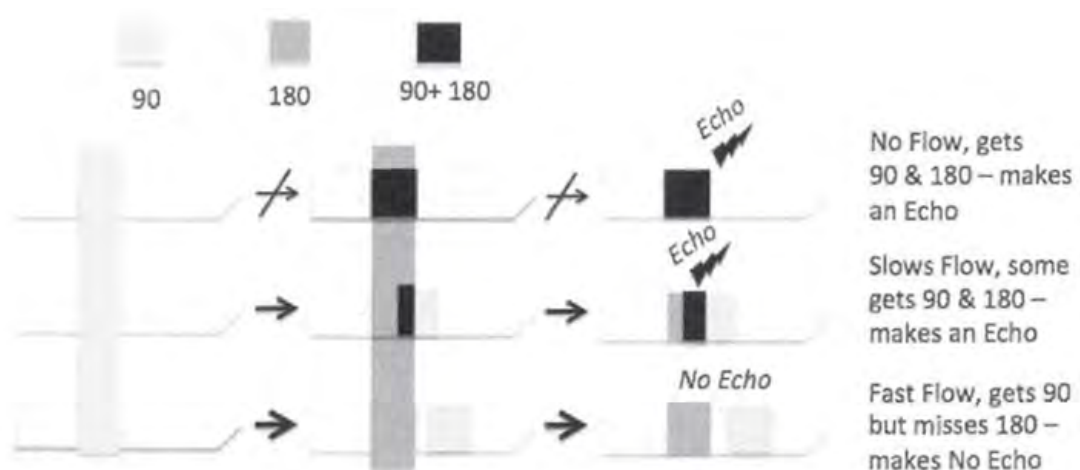
Making it better:

- Apply a saturation band adjacent to the imaging section - 90 pulse followed by crusher gradient.

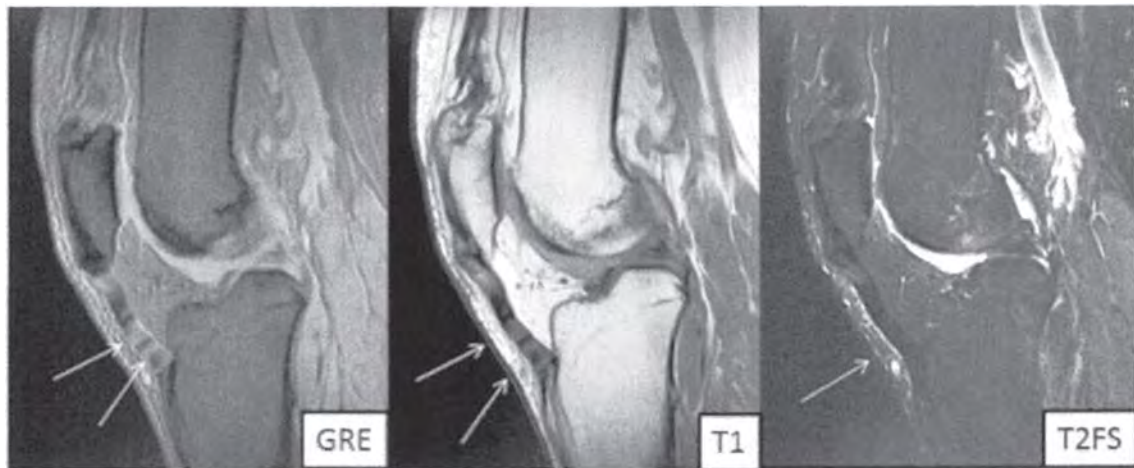
What Makes A Flow Void?

The dark hole of a patent vessel is called a “flow void.” This is generally a spin echo imaging finding (as stated above - flow looks dark on SE).

Why does this happen? To make an echo, the proton must be exposed to both a 90 pulse and a 180 pulse. If the blood is flowing fast it will get hit with the 90 degree, but then miss the 180 degree pulse. This means no echo, and thus no signal.



Magic Angle: This is an MSK artifact seen with tendons. You see this with short echo time (TE) sequences where the focus forms **an angle of 55 degrees** with the main magnetic field (magic angle phenomenon). This will NOT be seen in T2 sequences (with long TE). This phenomenon, is reduced at higher field strengths due to greater shortening of T2 relaxation times.



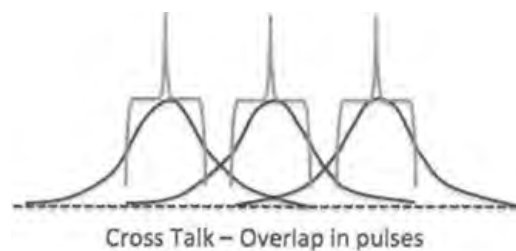
Magic Angle: You see it on short TE sequences (T1, PD, GRE). It goes away on T2.

RF Related: These include Cross talk and Zipper Artifacts

Cross Talk: RF and FT pulses are not perfectly rectangular. So if they are placed close enough together then you get excitation of neighboring section more than once in a single repetition. These lead to partial saturation and lower signal. So all sections (except the ones on the ends will be subjected to this).

Making it better: Increasing the gap between sections. Interleave slices (all odds, then all evens).

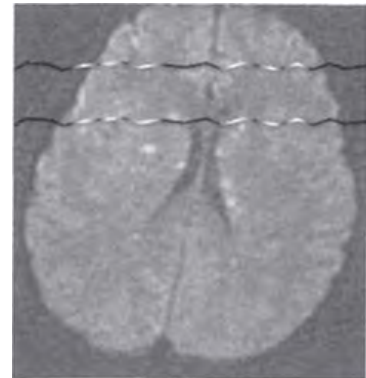
Testable Trivia: The 3D images are not susceptible to this artifact because the entire volume undergoes excitation with sections within the volume acquired with gradients.



Zipper Artifact: There are lots of random stray RF signals (radio, tv, etc...). Remember that the RF pulse is a “radio-wave.” Anyway, if you have defective or inadequate shielding you can get a “zipper” of high signal - 1-2 pixels in width running across the image. This typically extends in the phase encoding direction.

Gamesmanship: A possible scenario for this is that anesthesia left the pulse ox monitor in the room.

Making it better: Try closing the door (if it’s open). Remove all electronic devices from the room. Call the tech people to repair the faulty RF shielding. Alternatively, you could write an email, cc 15 people, and use the phrase “patient care”. That will ruffle some feathers.



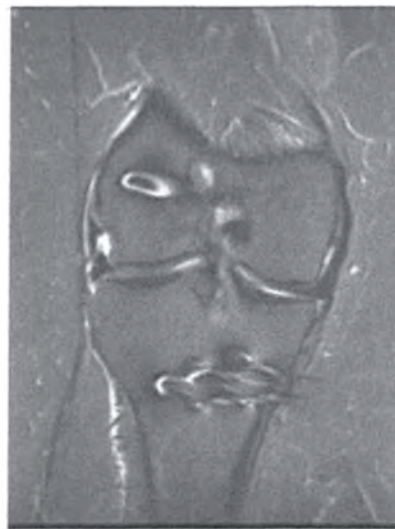
Zipper Artifact

External Field: This is magnetic inhomogeneity:

Inhomogeneous Fat Suppression: If the field is actually homogeneous you get a uniform fat saturation by applying an RF pulse with the resonance of fat protons. In the real world, local field inhomogeneities cause fat protons to precess at difference frequencies. These differences allow certain areas of fat to resist suppression - which can mimic edema.



T2FS: Fat Sat in bone marrow is ruined by metal messing with the local field



Improved with STIR Sequence

Making it better: Using an inversion recovery - STIR, especially in the setting of metal.

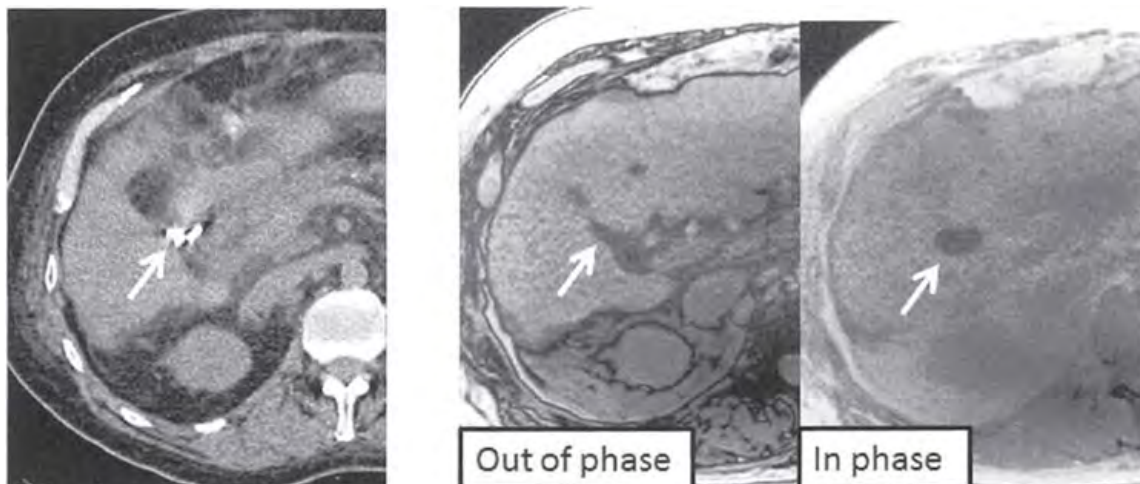
Magnetic Susceptibility: This includes diamagnetic, paramagnetic, and ferromagnetic

Susceptibility - This refers to the ability of a substance to become magnetized by the external field. Obviously metals have large susceptibility. Calcium hydroxyapatite and accumulations of gadolinium chelate can do the same thing.

Generally speaking, susceptibility affects all pulse sequences, but is most severe with GRE images and least severe with SE (because of the 180 degree refocusing pulse - to lose those T2* effects).

A less prominent version occurs at tissue interfaces (bone and muscle, or air and bone). The classic location for this is the transition from paranasal sinus to skull base.

Making it better: Using SE and FSE instead of GRE. You can swap the phase and frequency, use a wider receiver bandwidth, or align the longitudinal axis of a metal implant with the axis of the main field. STIR does way better than frequency selective fat suppression.



Notice the Blooming Artifact from the Metal Cholecystitis Worsens with Time (In-phase is done after Out-phase)

A key concept is that susceptibility artifact worsens on in phase imaging relative to out of phase. This has nothing to do with the phase of water and fat. Instead it has everything to do with in phase being done later. The longer TE, the more susceptibility. Remember, air will do the same thing.

Shimming

Shimming is a thing that can be done to improve field homogeneity. When the magnet comes from the manufacturer the field homogeneity is terrible. To correct for this a process called “shimming” is used. There are two types of shimming, passive and active. Most of the time a combination of the two is used; passive to get the thing going in the right direction and active to optimize the field for each patient.

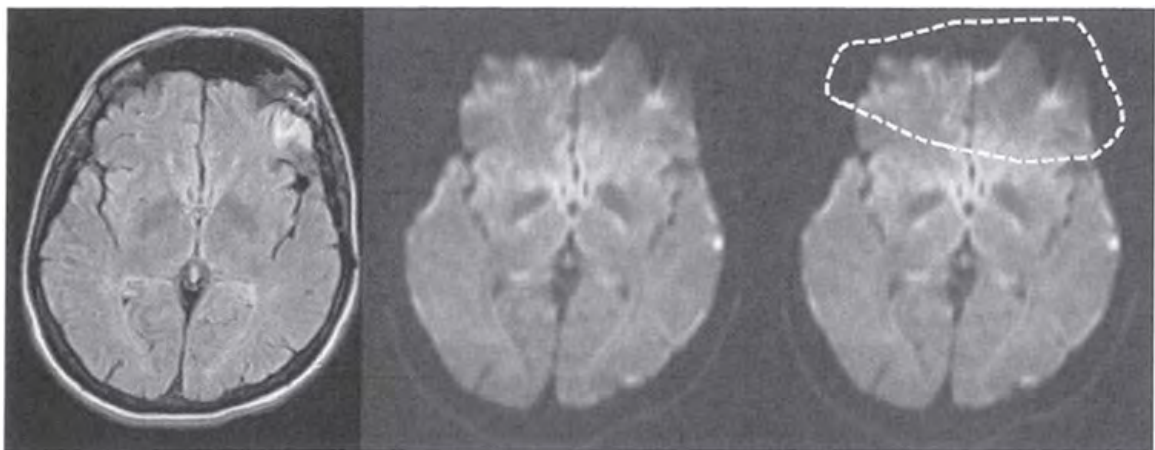
- * *Passive Shimming*- A phantom is scanned and the position of the shim plates is adjusted until the field becomes homogeneous (are at least better than it was). This is done at the time of installation.
- * *Active Shimming* - This is done by using the electromagnetic coil, and can be done after each patient (or sequence). Essentially, gives you the chance to have a homogeneous field (or nearly) regardless of the size of the patient.

Gradient Related: This includes Eddy Currents

Eddy Current: These things called “Eddy Currents” are generated when gradients are rapidly turned on and off. The actual location of said currents can be in the magnet, the cables, the wires or even in the patient. This looks like distortion (contraction or dilation of the image) or shift/shear.

Testable Trivia: Most severe with DWI pulses.

Making it better: Optimizing the sequence of gradient pulses.



FLAIR-for non-distorted comparison

Diffusion-Notice the stretch/ smear at the brain - bone interface. This is from Eddy Currents

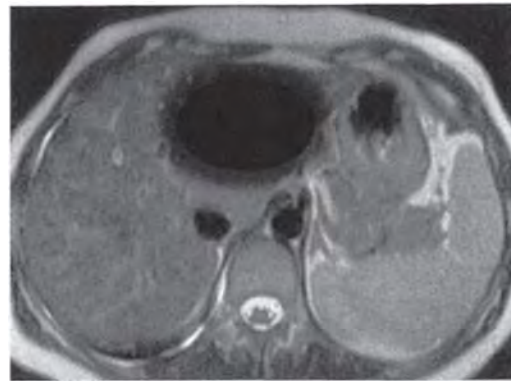
Errors in Data: This include dielectric effects and crisscross

Dielectric Effects / Standing Wave effects - Biologic tissues have a dielectric constant that results in reduction of wavelength by the inverse of some constant. Interactions can cause local eddy currents in the imaged tissues. Since RF waves are shorter at 3T - the effects are **worse with a stronger magnet**. You also see this worsen with large bellies, especially if they have **ascites**. Larger body parts (the abdomen) are primarily affected.

Classic Look /Location: Dark signal in the central abdomen over the left lobe of the liver.

Making it better:

- Application of dielectric pads - placed between patient and anterior body array coil
- Parallel RF transmission (SENSE) - RF pulses from a set of coils; each coil sends an independent RF pulse. Gives you a longer pulse.



Dielectric Artifact

Crisscross or Herringbone: If you see obliquely oriented stripes throughout the image, you are probably dealing with this artifact. The cause is data processing and/or reconstruction errors.

Making it better: Reconstruct the image again.

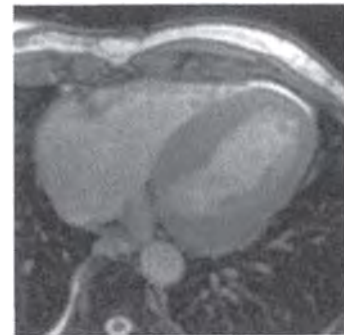
Artifact	Direction	Better	Worse	Trivia
Aliasing	Phase Encoding	<ul style="list-style-type: none"> •Increase the field of view •Change the phase encoding direction 	Smaller FOVs	Caused by a small FOV
Chemical Shift	Frequency Encoding	<ul style="list-style-type: none"> •Bigger Pixels •Fat Suppression •Increase Receiver Bandwidth 	<ul style="list-style-type: none"> •Stronger Magnetic Field •Lower Receiver Bandwidth 	Caused by differences in resonance frequencies
Gibbs/ Truncation	Both phase and frequency	<ul style="list-style-type: none"> •Bigger Matrix •Decrease Bandwidth •Decrease Pixel Size (increase PE Steps, Decrease FOV) 		<ul style="list-style-type: none"> •Caused by limited sample of FID •Classically seen in the spinal cord
Partial Volume		<ul style="list-style-type: none"> •Decrease Pixel Size (increase PE Steps, Decrease FOV) 	Thicker Slices	
Motion Artifact	Phase Encoding	<ul style="list-style-type: none"> •Saturation pulses •Respiratory gating •Faster sequences (BLADE, PROPELLER) 		
Cross Talk		<ul style="list-style-type: none"> •Increase slice gap •Interleave slices 		Caused by overlap of slices
Zipper	Phase Encoding			Caused by poor shielding
Field Inhomogeneity		•Shimming	GRE Sequences	Caused by geometric distortion
Susceptibility			GRE Sequences	<ul style="list-style-type: none"> •Caused by augmentation of magnetic field •Very bad in EPI
Eddy Current		•Optimize sequence of gradient pulses	DWI - large gradient changes	•Caused by geometric distortion or non-uniformity
Dielectric Effects		<ul style="list-style-type: none"> •Parallel Transmit •Use 1.5 T 	3 T	•Standing waves created as radiowave approaches length of body part
Magic Angle		•T2	T1, PD	•Occurs at 55 Degrees

Special Topic - Cardiac MRI

It's best to think of cardiac imaging in two flavors: (1) Bright Blood, and (2) Dark Blood.

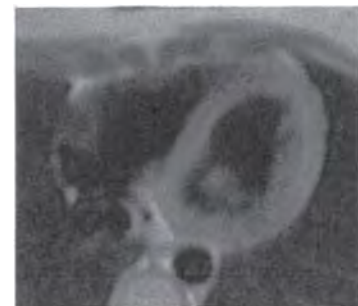
Bright Blood: These are gradient sequences, and they make up the majority of the cardiac work load.

Steady State Free Precession "SSFP" - This is a type of gradient sequence that is the primary sequence used for wall motion and volume analysis. If you see a CINE MRI of the heart it is probably a SSFP.



Gradient Echo – Bright Blood

Dark Blood: These are spin echo sequences. Remember that spin echo sequences have less susceptibility artifacts, so they can be of benefit when looking at the mediastinum in a patient who has had a prior sternotomy (with metal wires).

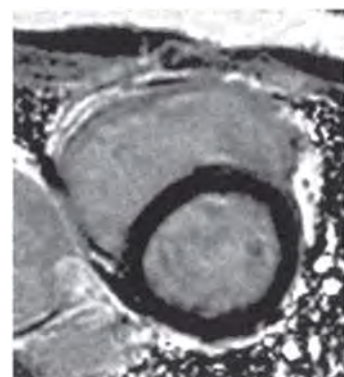


Spin Echo - Dark Blood

Inversion Recovery: Just like STIR nulls fat signal, and FLAIR nulls CSF signal, cardiac MRI uses an inversion technique to null myocardium. The reason you do this is to look for delayed enhancement (scar).

Trivia to know about inversion recovery:

A "PSIR" or Phase Sensitive Inversion Recovery or a T.I. Scout Series is done to help choose the correct time to use for inversion. This will produce a series of hearts. You want to pick the one with the darkest myocardium. This will give you the correct inversion recovery time.



Nulled Myocardium

Why not just do it like FLAIR or STIR, with a set time?
Although myocardium usually nulls at around 330 msec, it's highly variable depending on the person

Special Topic - Breast MRI

There are a few artifacts and technical differences specific to breast MRI that are worth knowing about. MRI of the breast is done for several reasons (high risk screening, implant rupture evaluation, surgical planning, etc...). The reason for the exam will dictate sequences.

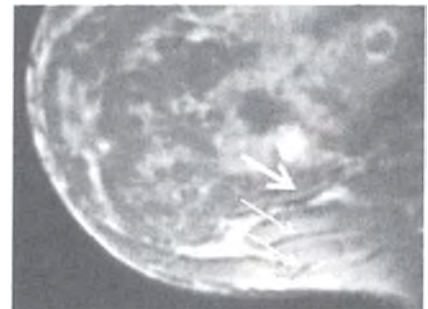
Testable Trivia: Breast CA screening will have dynamic post contrast sequences

Testable Trivia: Implant Rupture screening will NOT have contrast, but instead have a fat and water saturated sequence (only silicone will be bright).

Testable Trivia: A breast specific coil is used to increase SNR.

Artifacts:

Chemical Shift: You see this all the time in breast MRI, at the fat water interface. Remember this can be corrected by increasing the bandwidth to capture both fat and water in the same phase.



Chemical Shift Artifact

Motion / Breathing / Cardiac: Breathing and the beating of the heart would normally degrade the image on breast MRI. This is corrected for by running the phase encoding direction side to side, instead of front to back (like it is in body imaging).

Phase Encoding Direction:

Axial: Breast is left to right (Body Imaging is front to back)

Sagittal: Breast is top to bottom

Signal Flair - If the breast is too close to the coil element, it will not fat sat out correctly (look bright). You can fix this by repositioning the patient in the scanner.



Flair Artifact

Safety, Bioeffects, and FDA limits

Specific Absorption Rate (SAR)

SAR estimates the amount of energy deposited in a patient (*measurement of the rate the RF pulse dissipates in tissue*). The units of SAR are Watts/Kilogram (energy/mass).

Let's look at the formula - only for understanding some possible questions.

$$\text{SAR} = B_0^2 \times \text{Alpha}^2 \times \text{Duty Cycle}$$

Potential ways you could ask a question about this:

- (1) *If you double the B_0 , you do what to the SAR ?* It **Quadruples**
- (2) *If you double the Flip Angle (example 5 to 30), you do what to the SAR ?*
It **Quadruples**
- (3) *If you double the Duty Cycle (make the TR 1/2), you do what to the SAR?*
It **Doubles**
- (4) *Which has a higher SAR, Spin Echo or Gradient?* Spin Echo (they have higher flip angles - especially inversions).
- (5) *What are the limits?* Standards stipulate that no tissue shall endure a temperature increase of greater than 1 degree C. FDA limits are 4 W/kg over 15 minutes and 3 W/kg over 10 minutes.

Other Random MRI Safety Concerns:

- Static magnetic field (ferromagnetic materials): ferromagnetic materials present a risk in the presence of a strong magnetic field. Ferromagnetic objects, foreign or surgically placed, are at risk of displacement. Electronic devices such as cardiac pacemakers/defibrillators, nerve stimulators, etc, may suffer malfunction.
- ***Gradient Field (nerve stimulation)***
 - The rapid switching of magnetic field gradients can trigger peripheral nerve and muscular stimulation. EP1 sequences are most likely to cause such an effect, as they put the greatest strain on gradients.
- **Contrast agent safety issues**
 - The concern with Gadolinium contrast agents is Nephrogenic Systemic Fibrosis. The answer on the test is: GFR must be > 30 to administer Gadolinium.

Accreditation, quality control (QC) and quality improvement

- Weekly QC for accredited MR scanners (per ACR and performed by MR technologist): Center Frequency, table positioning, setup and scanning, geometric accuracy, high contrast resolution (verified by phantom), low contrast resolution, artifact analysis, film quality control, and visual checklist.
- Annual QC for accredited MR scanners (per ACR and performed by medical physicist or MR scientist) includes: Magnetic field homogeneity, slice position accuracy, slice thickness accuracy, radiofrequency coil check, display monitor check

You 're out of your element Donny

Section 10: Radiation Biology

“Exposure” - The ability of x-rays to ionize air, measured in Roentgens (R). This is the concentration, in air, of radiation at a specific point - and is the ionization produced in a specific volume of air.

“Absorbed Radiation Dose” or “Radiation Dose” - The amount of energy absorbed per unit mass at a specific point. It is measured in Gy or Rads (1 Gy = 100 rads). This is how much energy from ionizing radiation has been absorbed in a small volume.

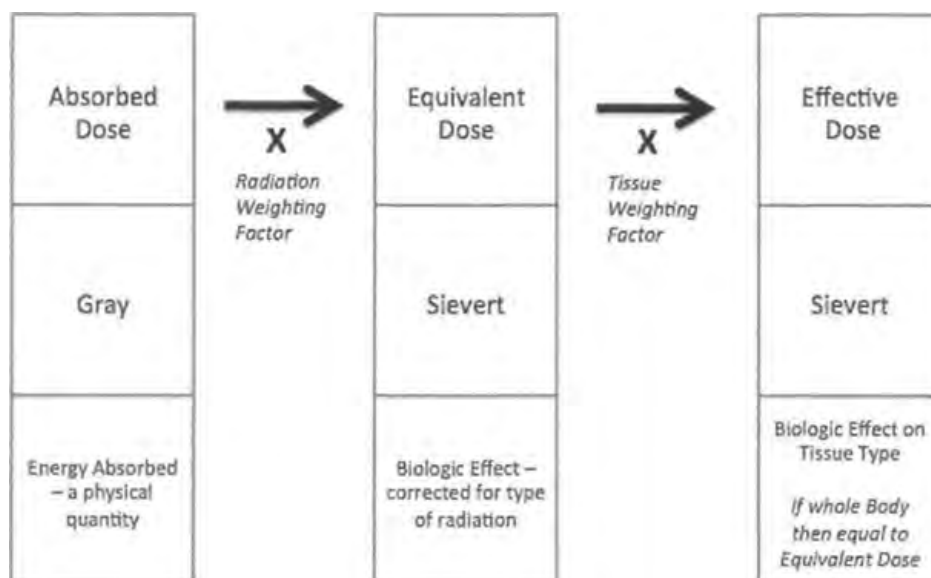
“Equivalent Dose” (EqD) - The absorbed dose of different types of radiation creates different levels of biologic damage (thus measured in Sv). A weighting factor is used to adjust the value. For example, an alpha particle can do more damage than an electron.

$$\text{EqD} = \text{Dose} \times \text{Weight Factor}$$

Weight Factor = for x-rays and gamma rays this is 1, for alpha particles it is 20.

“Effective Dose” (EfD) - This takes into account whether radiation has been absorbed by the specific tissue. In other words you are taking into account the type of radiation and the variable sensitivity of the organ / body part. If all the dose is absorbed then 1 Gy = 1 Sv. If you are dealing with organs or specific body parts you have to use a “tissue weighting” conversion factor.

$$\text{EfD} = \text{EqD} \times \text{Tissue Factor}$$



Kerma

Kerma is actually an acronym for “kinetic energy released per unit mass.” I want to take a minute and give a basic explanation of how energy is transferred. The process typically has two steps:

- (1) Energy is transferred to a charged particle (via Compton, P.E. effect, or whatever).
- (2) The now charged particle transfers energy to a medium via excitation and ionization.

Kerma is described step 1 (NOT step 2).

Why do I bother making this point?

- If you are dealing with a low energy photon, then Kerma is going to be the same as the absorbed dose.
- If you are dealing with a high energy photon, then Kerma is going to be MORE than the absorbed dose. This is because some of these secondary electrons are going to escape the area of interest before depositing their energy. These will be counted in Kerma, but not absorbed dose.
- In general, tissue doses are higher than air kerma (usually around 10%).

What is this “Air Kerma” ?

This is the sum of kinetic energy of all charged particles made when an x-ray (or gamma ray) passes through a unit mass of air.

What is this “Entrance Air Kerma” ?

This is the air where the x-ray beam would enter the patient (measured without the patient).

What is this “Air Kerma Product” (KAP) ?

This is supposed to account for the total amount of radiation on the patient. It is calculated by summing the Entrance Air Kerma x the Cross Sectional Area. The most important point is that KAP is independent of the source distance. This is because changes in beam intensity (from inverse square law) are matched exactly by changes in cross sectional area.

Trivia: SI Unit for Kerma = Gy

Stochastic Effects vs Deterministic Effects:

Deterministic Effects	Stochastic Effects (“Random”)
Has a threshold	Has NO threshold
Severity is dose related	Severity is NOT dose related
	Probability of effect increases with dose
Does Not include Cancer Risk	Includes heritable effects and carcinogenesis (NOT cell killing)

Interaction of Radiation with Tissue:

As discussed before you have two primary methods that x-rays interact with matter; the photoelectric effect and Compton scatter. The key interaction is the production of a free electron, which can then transfer its energy to other electrons by ionization and excitation. The actual **damage done is the result of ionization** produced by x-rays / gamma ray photons giving energy to orbital electrons and alpha / beta particles interacting electromagnetically with orbital electrons.

What Determines the Biologic Effects from Ionizing Radiation?

Primary variables include those inherent to the cells and the conditions of the cells at the time of irradiation. There are also variables related to the radiation (absorbed dose, dose rate, type of radiation, and energy of radiation).

Damage to biologic systems occurs in this order:

Molecular -> Cellular -> Organism

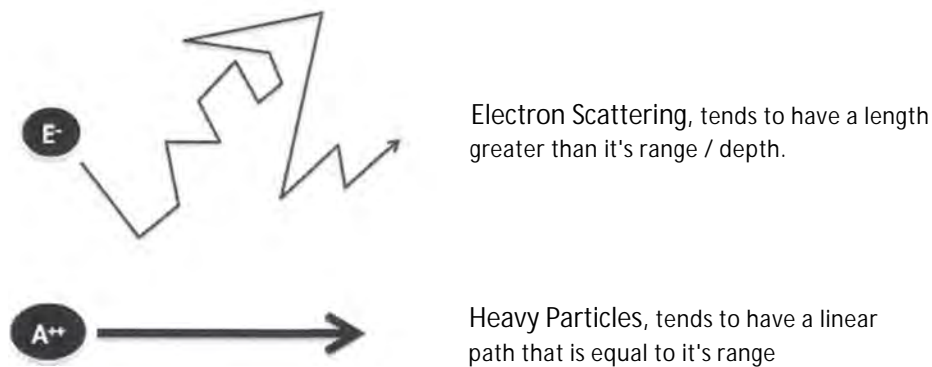
Molecular damage is always first. Ionized atoms do not bind properly to other molecules. Loss of function in the molecule leads to loss of cellular function.

Charged Particle Tracks:

When radiation crashes into a biologic material, energy is deposited along the track. The pattern of this deposited energy depends on the type of radiation involved.

X-Rays / Gamma Rays: X-ray ionization density is low along the track

Neutrons, Protons, and Alpha Particles: Ionization events occur much more frequently.



Concepts / Vocabulary:

Linear Energy Transfer (LET) - This is the **average amount of energy deposited per unit path** length of the incident radiation. LET is important for assessing the potential tissue and organ damage. LET comes in two flavors:

- **High LET:** Neutrons, Protons, Alpha Particles, and Heavy Ions. These are MUCH MORE DAMAGING ("has a higher quality factor").
- **Low LET:** Photons, Gamma Rays, Electrons, and Positrons

Relative Biologic Effectiveness (RBE) - This is the relative capability of radiation with differing LETs to produce a particular biologic reaction.

$$\text{RBE} = \text{Dose of 250 kV x-rays} / \text{Dose in Gy of Test Radiation}$$

Example Problem: A reaction is produced by 5 Gy of test radiation. It takes 10 Gy of 250 kVp x-rays to produce the same reaction. What is the RBE ?

$10 / 5 = 2$; In this example the test radiation is 2 times as effective in producing this biologic reaction than the "standard" 250 kVp x-rays.

The “Kill Effect”

As LET increases, RBE will increase... to a certain point.

Above 100 keV/micrometer of tissue, RBE decreases with increasing LET - because the maximum potential damage has already been done - additional increase in LET is wasted dose.

Oxygen Enhancement Ratio (OER) - This is the relative effectiveness of radiation to produce damage at different oxygen levels. The idea is that biologic tissue is more sensitive to radiation in an oxygenated state. A testable piece of trivia is that OER really only matters for low LET radiation. With high LET radiation the OER is often 1 (biologic damage without oxygen = biologic damage with oxygen).

Direct vs Indirect Ionizing Radiation: Actions, are called “direct” if they act “directly on the DNA.” Actions are called “indirect” if they act on water. **The majority of irradiation in living cells is the result of indirect action on water** (your body is 70% water and < 1% DNA). This indirect action on water creates a free radical which then jacks the DNA.

<i>This vs That:</i> Direct vs Indirect Radiation			
Direct Radiation (minority)	Acts on DNA	Most likely for High LET Radiation (unusual in x-ray imaging)	
Indirect Radiation (majority)	Acts on water in the cytoplasm, creating free radicals - which in turn damage the DNA	More likely for Low LET Radiation	This process is promoted by the presence of oxygen

Effects on DNA

Single Strand Break - Ionizing radiation can cause a break in one of the chemical bonds (point mutation). This is more likely with low - LET radiation. Repair enzymes can move in and fix this.

Double Strand Break - Ionizing radiation can cause a break in multiple chemical bonds. These are more likely with high - LET radiation. These are harder to fix.

Mutation - It's possible for radiation to cause a loss of or change in the nitrogenous base in the DNA chain. If the cell doesn't die from this, this incorrect information will be transferred as the cell divides.

Testable Point: The syndrome with the most sensitivity to x-rays is Ataxia Telangiectasia. Common distractors include Bloom Syndrome and Fanconi Anemia (both of which have genetic instability but no particular relationship with x-rays). Xeroderma pigmentosa is another classic distractor - but it is more sensitive to UV radiation.

Chromosome Anomalies - Two types have been described at metaphase.

Chromosome Aberrations - Damage occurs early in interphase (before DNA synthesis). Both chromatids are broken so each daughter will get a broken copy.

Chromatid Aberrations - Damage occurs later in interphase (after DNA synthesis). Only 1 chromatid is going to have a break (the other one will be fine)

Effect on the Cell:

Testable Point - An x-ray or gamma ray dose of 1000 Gy in a period of second / minutes will cause instant death of a large number of cells.

"Mitotic Death " - This is when a cell dies after 1 or more divisions. A relatively small dose of radiation can cause this.

"Mitotic Delay" - A very small dose (0.01 Gy) just before a cell starts to divide can cause a delay or failure in the timing of the normal dividing.

Cell Cycle Phase - Cells are MOST sensitive during M phase (mitosis). They are more sensitive during late G2 phase. They are less sensitive during G1 phase. They are the LEAST sensitive during late S phase.

Testable Point: In order of sensitivity: $M > G2 > G1 > S$.

Testable Point: G1 is the part of the cell cycle that is the most variable in length (shorter in cells that turn over quickly).

Surviving Cell Synchronization - If you blast a group of cells with radiation, the ones that survive will have their cell cycles synchronized. The reason is that the most resistant part of the cell cycle is late synthesis. So most of the surviving cells are in this phase.

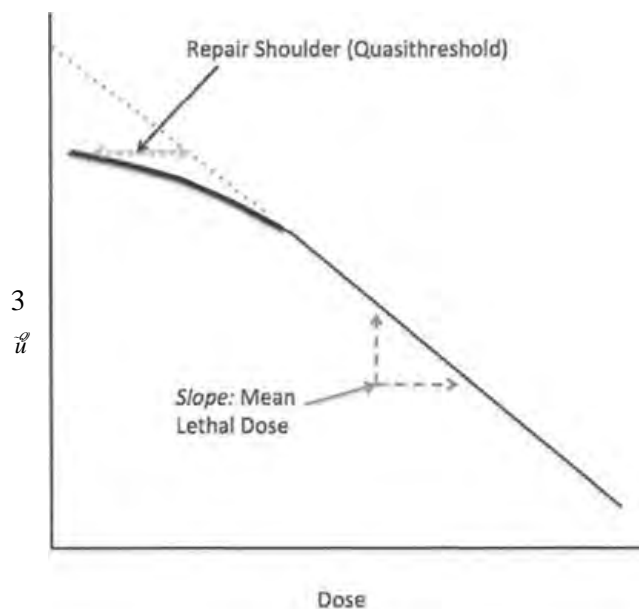
Law of Bergonie and Tribondeau - Cell sensitivity is directly related to their reproductive activity, and inversely related to their differentiation. So, the more a cell turns over (skin, blood, GI tract lining, reproductive cells) the more sensitive they are. The less cells turn over and more differentiated they are (brain, nerves, muscles) the less sensitive they are.

Testable Point: The small bowel is the most radiosensitive part of the GI tract.

The Survival Curve (possible source of questions).

This is a graph of dose vs cell survival. The point to notice is the **“Quasi-threshold dose”** which is the portion of the graph when repair mechanisms are in there trying to hold the ship together. It is a measure of “sub-lethal” damage to the cell.

A testable point is that this shoulder only exists with low-LET radiation curves.



1 / Slope (D_0) describes the linear portion of the curve, and the radio-sensitivity of the cell population. The higher the D_0 the more radio-resistant the cell is.

Key Points:

- Higher Dose Rate makes a smaller shoulder, and steeper drop in the curve
- Low LET will have a shoulder, High LET will NOT have a shoulder
- Oxygen will make a steeper drop in the curve (more pronounced with low LET)

Effect of Ionizing Radiation of Blood:

Lymphocytes are the MOST sensitive blood cells in the body. A dose of 0.25 Gy is enough to depress the amount circulating in the blood.

Lymphocytes can actually be stimulated to divide and you can score the number of chromosomal aberrations at first mitosis to reflect average total body dose.

Acute Radiation Syndrome (ARS):

This is a clinical response when the body is hit with a large amount of radiation. There are 3 subtypes of this syndrome. You typically have 4 phases. You start out feeling terrible (GI flu like symptoms - nausea / vomiting / diarrhea), then you feel better “latent phase,” then your syndrome subtype manifests - related to underlying organ system as below, the fourth phase is death/recovery.

Acute Radiation Syndrome	Dose Needed	Latent Period	Outcome
Bone Marrow	>2 Gy	1 -6 Weeks	You do worse with higher doses. It's possible to survive.
GI	> 8 Gy	5-7 Days	Death within 2 weeks
CNS	> 20-50 Gy	4-6 Hours	Death within 3 days (<i>unless you get to Elysium</i>)

Testable Point: A total body dose of 0.75 -1.25 Gy will cause nausea about 30% of the time.

For Prospective:

mGy = A dose that is diagnostic. For example, 30mGy is a dose for a CT Abd and Pelvis.

Gy = A dose that makes you sick. For example, 2 Gy is going to mess with your bone marrow.

Triaging Patients with Possible ARS:

This basic idea is that the earlier the symptoms appear the worse they are going to do.

GI	Skin	WB Dose (Gy)	Action
No vomiting	No skin redness	< 1	Surveillance for 5 weeks
Vomiting 2-3 hours after exposure	Skin redness (12-24 hours after exposure)	1-2	Surveillance for 3 weeks, Consider General Hospital
Vomiting 1-2 hours after exposure	Skin redness (8-15 hours after exposure)	2-4	Hospitalize - Bum Center
Vomiting < 1 hour after exposure	Skin redness (1-6 hours after exposure)	>4	Hospitalize - Specialized Radiation Center

High Yield Point: Early Vomiting is a marker of severity / poor prognosis.

Effect of Ionizing Radiation - Lethal Dose 50/30:

This is the dose which will kill 50% of people within 30 days. The LD 50/30 for a person is around 3-4 Gy without treatment. With medical treatment you may be able to tolerate up to 8.5 Gy (*depending on if you ate your wheaties*).

Lethal Dose 50/60 = Lethal dose at 60 days for 50% of the population. This is used for bone marrow failure. This is about 3-4 Gy.

Lethal Dose 50/4 = Lethal dose at 4 days for 50% of the population. This is used for GI failure. This is about 10 Gy.

Genetically Significant Dose (GSD) = This term is used for expressing genetic risk as an index of presumed impact on the entire population. GSD is the dose if received by all members of the population that would result in the same hereditary damage as the actual doses received by the gonads of the people who actually get radiation exposure. This depends on gonadal dose, and child bearing potential (age of patient, sex of patient).

Increased Risk of Cancer: The risk of cancer (estimated by BEIR 5 and UNSCEAR) was 8% / Sv. A reduction factor of 2 is used for low dose and low rate - so the working **population has a risk more like 4%-5% /Sv**. Some people throw around the following calculation:

Risk for Hereditary Effect of Radiation:

$$0.2 / 100 \times \text{Dose}$$

Example: Women gets CT scan 0.3 Gy then gets pregnant 1 year later. Chance of radiation induced hereditary defect? $0.2 / 100 \times 0.3 = 6 \times 10^{-4}$ or 6 in 10,000

Latency: The time between exposure and appearance of cancer is sometimes called the latency. It's variable for cancers. For example, leukemia has a short latency of 5-7 years, where as solid tumors have a latency as long as 20-50.

Radiation Effect on a Fetus: The big testable point is that 2 Gy gets you an abortion during the first two weeks (all or nothing). 2 Gy gets you congenital abnormalities during week 2-6 (period of organogenesis). Between weeks 8-15 you can get reduced head diameter and mental retardation. IQ is said to drop 30 points per 1 Sv, with the risk of retardation being 40% at 1 Sv. It takes a very low dose (just a few radiographs) to the fetus to increase the risk of childhood leukemia.

Testable Point: The fetal thyroid does NOT take up iodine prior to week 8. So, if mom gets ¹³¹I prior to week 8 the fetus will not be hypothyroid (after that it's hosed).

Skin Problem	Dose (Gy)	Onset
Early Transient Erythema	2 Gy skin dose	Hours
Severe “Robust” Erythema	6 Gy skin dose	1 Week
Telangiectasia	10 Gy skin dose	52 Weeks
Dry Desquamation	13 Gy skin dose	4 Weeks
Moist Desquamation / Ulceration	18 Gy skin dose	4 Weeks
Secondary Ulceration	24 Gy skin dose	> 6 weeks

Hair Problem	Dose (Gy)	Onset
Temporary Epilation	3 Gy	21 Days
Permanent Epilation	7 Gy	21 Days

**/ remember this by saying $3 \times 7 = 21$*

Symptom / Issue	Dose
Nausea (30% of people)	0.75-1.25 Gy WB
Depress circulating lymphocytes	0.25 Gy WB
LD 50/60 (Marrow)	3-4 Gy WB
LD 50/4 (GI)	8-10 Gy WB
LD for CNS	>20 Gy (20-100) WB
Double the natural or spontaneous mutation rate	1 Gy
Effective dose from background radiation in the US	3 mSv per year

Sterility / Infertility

Females: The threshold is age dependent. The younger the patient, the more dose they need. Close to puberty think about 10 Gy, Close to Menopause think about 2 Gy.

Male: Temporary sterility is going to occur somewhere between 0.15- 2.5 Gy. More permanent sterility requires an acute dose around 5 Gy.

Sterility / Infertility

Male Temporary	0.15-2.5 Gy
Male Permanent	5 Gy
Female Age 12	10 Gy
Female Age 45	2 Gy
Female - No age given	Just say around 6 Gy

Cataracts

Trivia: Senile cataracts tend to involve the anterior lens. Radiation induced cataracts tend to involve the posterior lens.

Trivia: 1R people tend to be the ones to get these... sorta makes sense.

The cataract tends to develop years (like 20 years) after exposure. This latent period is inverse to the exposure amount.

The threshold for development from an acute exposure is around 2.5 Gy (some studies go as low as 0.5 Gy.)

Cataract

Acute Exposure Threshold to Cause Cataract	2.5 Gy
Annual Dose Rate Limit	0.15 Gy/Yr

Exposure Limits	
<i>Occupational Workers (minimal age 18)</i>	
Lens	150 mSv/year (some new papers say 20 mSv)
Radiation Worker	50 mSv/year
Extremity	500 mSv/year
<i>Public Exposure</i>	
Infrequent	5 mSv /year
Continuous	1 mSv/year
Embryo/fetus	5 mSv .year
Embryo Fetus (post declared pregnancy)	0.5 mSv/month
Controlled Areas	50 mSv/year
Uncontrolled area	5 mSv year
Genetically Significant Dose	0.25 mSv
Effective dose from background radiation in the US	3 mSv per year

MRI Safety Trivia:

Specific Absorption Rate (SAR)

The RF power absorbed per unit mass.

Depends on number of images acquired per unit time, patient dimensions, RF waveform, tip angle, and coil type

The SAR should not exceed 4W/kg for the whole body for 15 mins.

RF heating is considered acceptable if the core temperature increase is $< 1^\circ\text{C}$.

Noise: The noise with MRI is the result of vibrations in the gradient coils. The peak acoustic noise should not exceed 140 dB.

Bunts: If you have a “conductive loop” - either from crossed arms, ECG leads, or unconnected surface coil leads in contact with skin it’s possible to get a nice thermal injury (and a law suit). If the patient is contacting the inner bore (where high level standing RF waves are highest) there can be a bum (and a law suit). Implanted metal objects can also heat up.

Peripheral Nerve Stimulation: Rapid changing of gradients has the potential to cause peripheral nerve stimulation, and muscle movement.

NSF (Nephrogenic Systemic Fibrosis) - This is a very serious, extremely rare (possible not real) thing that caused fibrosis of skin, joints, and organs. It was seen in end stage renal failure patients ($\text{GFR} < 30$), who got Gd. The risk is related to the chelation structure, not the actual Gd.

- Most Likely To Cause - “Linear Non-Ionic” - Omniscan , Optimark
- Intermediate Risk - “Linear Ionic” - Multihance, Magnevist
- Least Likely to Cause (most safe) - “Cyclic Structure” - Gadovist

Pregnancy - There is NO evidence that MRI is dangerous to the fetus. Having said that, most institutes tend to say no contrast in pregnancy. * **Fear of Litigation > Rational Thought*

High Yield Radiation Biology/Safety Blitz:

- The majority of energy received by biologic material from x-rays is transferred by electrons
- Two thirds (around 60%) of x-ray damage to biologic material is mediated by free radicals
- Interaction of two separate chromosomal breaks can lead to aberrations such as dicentric and rings
- Double stranded DNA breaks are the most important lesions caused by x-rays.
- The final number of double stranded DNA breaks is more important than the initial number of breaks - because some will be repaired.
- You can score whole body radiation exposure by stimulating lymphocytes to divide.
- Risk of Radiation induced CA: 4-5% per Sv = Adult, Up to 15% per Sv for Child , About 1/10th that for someone older than 50
- Transient skin erythema can be seen in hours, with the main wave occurring after 10 days
- Radiation induced sterility in males does NOT affect hormone levels or libido.
- Radiation induced sterility in males has a “latent period” between irradiation and sterility
- Radiation induced sterility in females causes symptoms similar to menopause.
- Carcinogenesis by radiation is stochastic (all or nothing) - based on Beir 5 and UNSCEAR committees
- Sentinel Event = 15 Gy
- The thyroid can have radiation induced benign and malignant nodules
- Radon workers get more lung cancer
- Cataracts caused by radiation start in the back of the lens
- If Al-Qaeda attacks your hospital with a dirty bomb, remember that the casualties may present a risk to medical personnel (because they are contaminated with radionuclides).
- Inhaled Radon (an alpha emitter) contributes about 55% of the effective dose to the US population, and is the largest single contributor to effective dose.
- The greatest source of exposure to ionizing radiation for the general population of the United States from *due to human activity* is medical imaging.
- For MRI; you must have a controlled access so that the fringe field outside the area doesn't exceed 5 Gauss.
- For MRI; there is a thing called “Specific Absorption Ratio” - which is the RF power absorbed per unit mass. The SAR should not exceed 4W/kg for the whole body for 15 mins.

You're killing your father, Larry!

16

Non-Interpretive

Prometheus Lionhart, M.D.



As stated in public interviews, the idea for this portion of the exam is for the 3rd year resident to show that he/she can do more than read studies - but can also “serve and lead.” Topics for this chapter were taken from the study guide published on the ABR’s website, and the articles references within said study guide. To view that masterpiece for yourself, simply google “ABR non-interpretive skills study guide.”

Section 1: “Quality” & Other Random Meaningless Buzzwords

QA vs QI

Back in the dark ages Doctors had “QA” or “Quality Assurance” meetings. This is of course a laughable practice that is backward and outdated. “QA meetings” are like using leaches to cure an imbalance of the bodily humors. Now, in the modern age, Doctors (or people who wished they went to business school instead of medical school) use the term “QI” or Quality Improvement. This change in vocabulary has really revolutionized medicine.

QA- old term : reactive, retrospective, punitive or finger pointing.

QI- new term : prospective and retrospective reviews; aimed at improvement.

The “I” in QI stands for Improvement. If you want to improve your rank in academic institutions you better learn about QI. This is how you make full professor, so pay close attention.

Various Catchphrases and Buzzwords Invented By Career Administrators

Best Practices: The idea behind “best practices” is that everyone should do things the same way. The “best” way. Of course, if everyone does it that way it just makes it a “mediocre practice.”

Dashboard - A visual display of the most valuable information you need to achieve your objective.

Benchmarking - Measuring the quality of organizational policies by comparing them with standard measurements or peers.

TQM: Total Quality Management - When reading about TQM my favorite line is this “there is no widespread agreement as to what TQM is and what actions it requires of organizations.” In other words, it’s just a made up buzzword. Having said that, I’d remember that quality improvement should be continuous, with responsibility in the top management, and that the definition of quality is defined by what customers require.

CQI: Continuous Quality Improvement - Another meaningless buzzword that is essentially the same thing as TQM.

Key Performance Indicators “KPI”: Ideally this is a reproducible measurable thing - like patient safety, customer service satisfaction, or the number of publications a faculty member has. This tool can be used to assess the well-being of the department and need for potential improvement / intervention.

Methods to Improve Quality:

PDSA (Plan-Do-Study Act)

This is a powerful, amazing, and totally original tool that can be used for an action-oriented process. The idea is that you chain the 4 steps together

- * **Step 1 “Plan”** : What needs fixed and how should we fix it.
- **Step 2 “Do”** : Do the thing, and record data.
- * **Step 3 “Study”** : Did it work? Or Not work?
- **Step 4 “Act”** : If you failed in step 3, then *fix the root cause* and implement a new plan (repeat at step 1).

Lean:

“Lean” is a style of organizing workflow that has two main principals:

- (1) Elimination of Waste through standardized workflow (*no unnecessary variation*).
- (2) Respect for Long Time Employees, and Customers

There are 4 “tools” that are used to create a Lean workflow. *This is critical knowledge for being an intermediate level radiologist.

- **Value Stream Mapping:** This is a tool to help improve workflow, by creating a “visual map” of the entire process from beginning to end. You can create alternative maps of how things could be done differently for QI projects.
- **Five S:** This is the kind of thing that gets you promoted straight to the top. Take 5 words that all mean something similar and start with the same letter, and give it a catchy name “FIVE SI.” Boom , instant promotion. In this case, the 5 words basically mean “organized” or “standardized.”
- **Pull Systems:** This is the idea that you don’t overwork one guy in the assembly line. You create a system where work flow is constant. Never have one guy making 3 pieces in step 2, while the guy at step 3 only makes one piece. That way no one is sitting there idly. You don’t pay people to idle. You want to be Chairman? You gotta grind every last drop of sweat and blood out of your trainees.
- **Error-Proofing:** The idea that workers should draw immediate attention to defects, so that the bosses can come in and fix them.

Design-Measure Analyze Improve Control (Six Sigma)

Sigma = Standard Deviation. The idea is to target an error rate of 6 standard deviations from the average.

01 Tools *(Overview)*

The Big 5 *(expanded discussion with examples to follow)*

- * Brainstorming - Generating a big list of ideas
- * Fishbone Diagram (Cause and Effect): Categorize and organize contributing factors, in a cause and effect pathway.
- * Flow Charts: A graphical map to show the steps / decision points involved in a process. There are two subtypes. “High Level” which describes a process from beginning to end. “Low Level” contains more details about the major steps.
- * Pareto Charts: Lots of things are involved in the creation of a problem, but only some of them contribute in a manner that is actually responsible. In other words, changing one thing might fix a problem, even though 10 things contributed to it.
- * Shewhart Charts (Control Charts): Is the process stable? Do we need to do a formal examination for quality? These are the questions a control chart (Shewhart) answers. The graph is generated by placing success on the numerator, and total opportunities on the denominator. The graph shows the changing process over time.

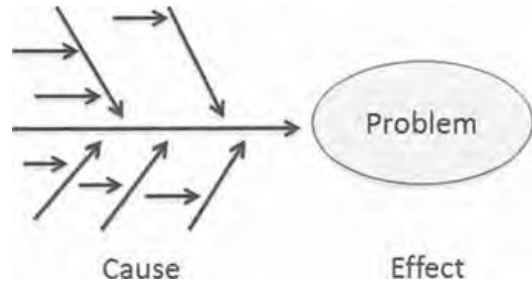
Other Tools *(still testable, but less high yield)*

- * Multivoting - People choose the highest priority problem. Based totally on popular opinion.
- * Nominal Group Technique (NGT) - Used to deal with a big mouth in the room. Minimal conversation or interaction is tolerated. The technique has two stages: (1) Brainstorming by asking a question and having people write down their response. (2) Ideas are ranked by individuals, with the scores of the groups used to decide what is good.
- * Prioritization Matrix - Things are ranked by specific criteria (how fixable, how serious, etc...). A matrix totaling all votes is then created.
- * Walkthrough - Trying to spend a day in the shoes of your underling (or a patient). See if the problems are real, or bottlenecks in work flow can be identified.

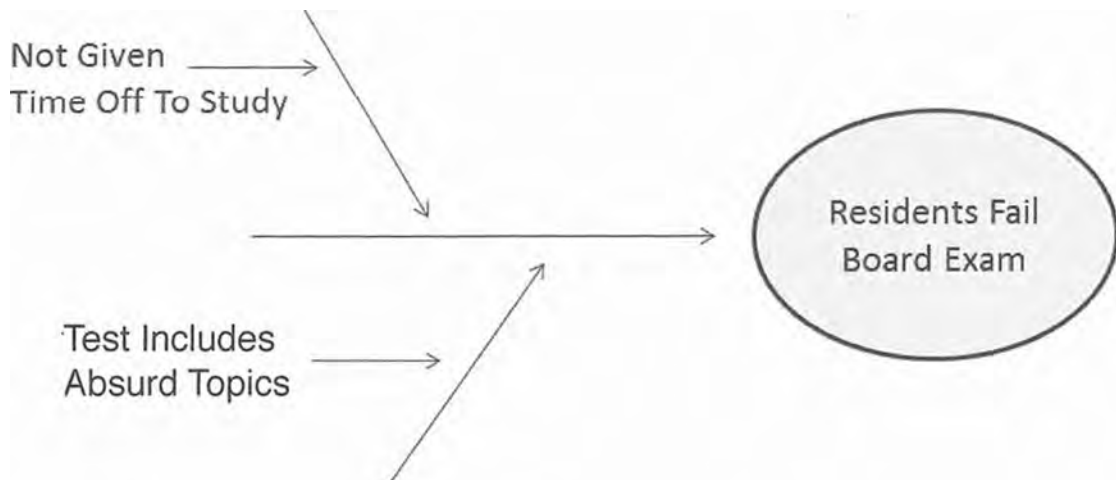
Tool	“Catch Phrase” / “Buzzword”
Brainstorming	Identify all the Issues
Fishbone Diagram	Cause and Effect
Flow Charts	“Clarify the steps and decision points”
Pareto Charts	80% of problem, explained by 20% of causes
Shewhart Charts	Is the process “under control” or stable?
Multivoting	People’s opinion of what is most important
Nominal Group Technique (NGT)	No Big Mouths. Minimal talking, lots of idea writing.
Prioritization Matrix	Rank then and add them up

Fishbone Diagram (Cause and Effect):

This type of chart is used to organize the ideas that contribute to portions of a problem or process. The process of drawing one is essentially asking the question “cause and effect” over and over and over again.

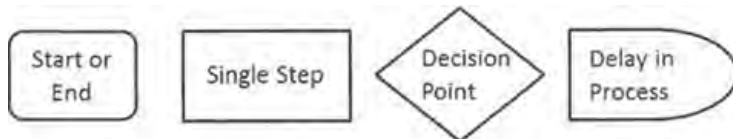


Example:



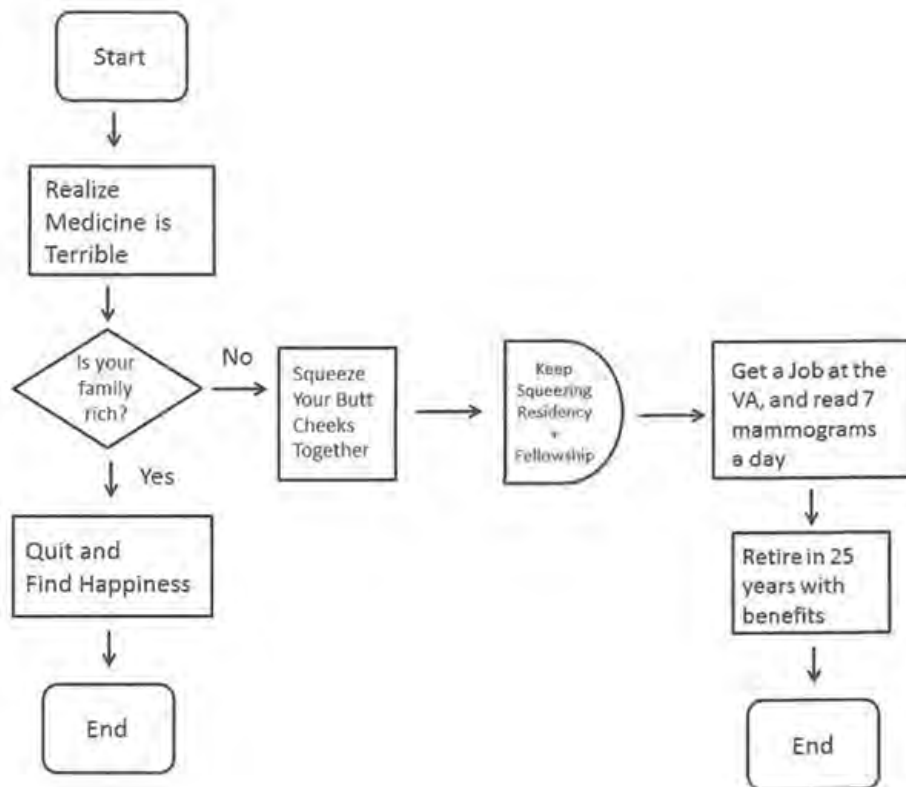
Flow Chart:

Flow charts use symbols chained together to illustrate a process. The standardized shapes for the major symbols are below.



Processes can be high level (“get up go to work”), mid level (“get up, get in shower, eat, drive to work”), or low level (“get up, walk into bathroom, pee in toilet, flush toilet, start shower water”).

Example:



Testable Trivia:

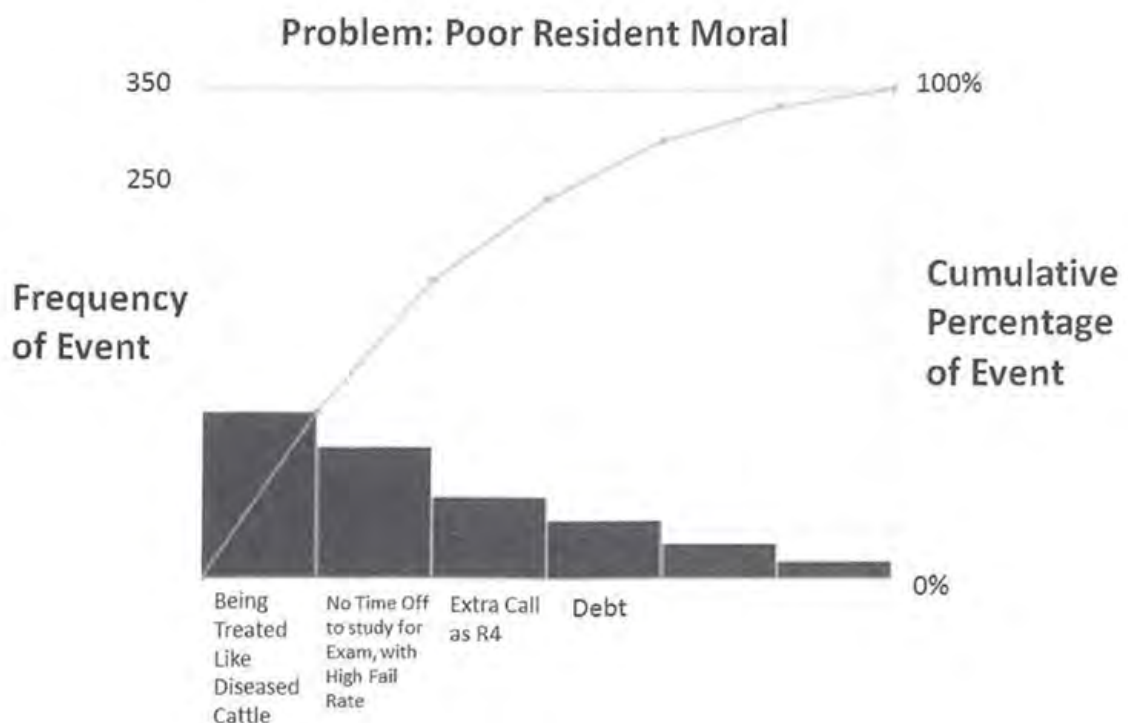
- * Flow charts are good for “cause and effect”
- * Only use one arrow for yes, and one arrow for no.
- * Draw the boxes first, then the lines
- * It’s OK to move the start and end boxes, after you start.

Pareto Charts

This is a chart named after Vifredo Pareto, who accomplished many great things in his life including popularizing the tenn “elite” , marrying a women 30 years his junior (WIN!), and collecting a large number of cats - which he famously treated better than his house guests (he fed the cats first, and made his dinner guests wait). Oh, he also made a chart.

The chart contains both a line graph and a bar graph. The bars are ranked in descending order, creating a concave function. The entire **purpose of the chart is to highlight the most important factor among a set of factors**. The idea is that fixing the main cause, may fix the problem.

Example:



Shewhart Charts (Control Chart)

This is a “process-behavior” type of chart. The idea is to be able to look and see if a process is under control (with just stable variation), or not under control. Upper and lower “controls” are chosen based on the definition of success and failure for the system. Staying between the controls demonstrates a stable process.

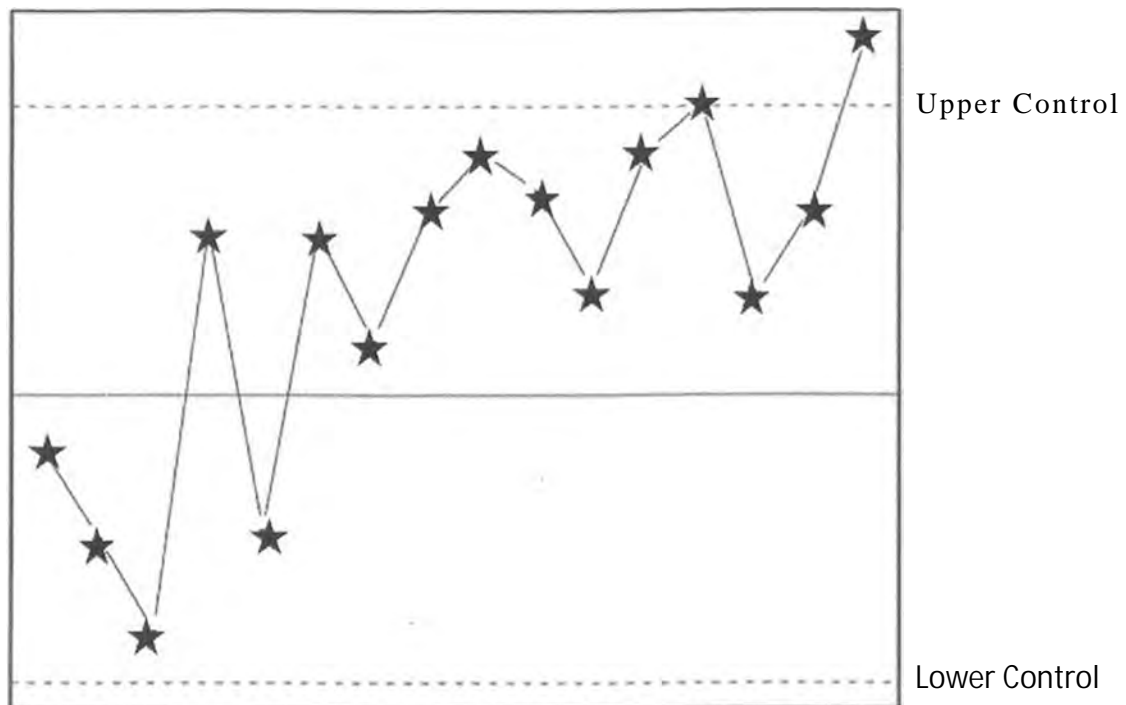
Testable Trivia:

What the chart looks like

What is the numerator? Samples of Success

What is the denominator? Total Opportunities

Example:



Section 2: MRI and CT Safety

MRI “Zones”:

What are the 4 zones?

Zone I: No restriction. This is basically outside the building.

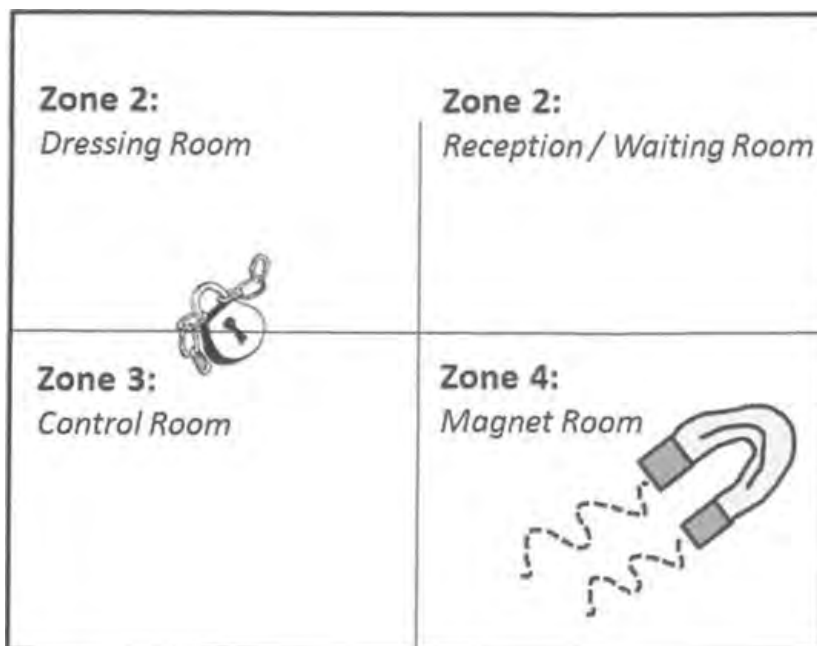
Zone II: No restriction. This is the waiting room and the dressing room. This is where you can screen patients and control access to Zone 3 and 4.

Zone III: Restricted Room. This is typically the control room, where the MRI tech does his/her thing. There should be some kind of a lock on the door between zone 2 and 3.

Zone IV: Restricted Room. This is the actual MRI scanner room (the same room as the magnet).

Zone 1:

Outside the Building



Zone Trivia:

Are there any exceptions for entry into restricted access zones? Access is restricted to zone 3 and 4 (without filling out the screening forms ect.. .) **There are NO exceptions to the guidelines restricting entry to zone 3 and 4.** Even if the patient codes in the scanner there are no exceptions (*per the Kanal article cited below*). The MRI techs should start CPR on the patient in the room (zone 4), stabilize the patient then get them out of zone 3 and 4 to a holding area where the code team can actually save them.

If there is a code in the scanner should you quench the magnet? Typically not. The magnetic field will still be around for a minute or two. Plus, that is really expensive and the bean counters who run the hospital will be pissed. Instead, do CPR then bring them out of Zone 3 and 4.

MRI Screening begins with? A focused history to identify patients who may have metal in them.

What if the Patient is insane, unconscious, or has terrible body odor and you can 't initiate the screening process with him/her ? You will need to ask family members, consult the medical record, and possibly do screening x-rays (orbits etc...) as needed.

Kanal, Emanuel, et al. "ACR guidance document for safe MR practices: 2007. "American Journal of Roentgenology 188.6 (2007): 1447-1474.

CT Contrast - Reactions and Management:

Statistical Trivia - How Often Do Reactions Occur?

- Incidence of Any Contrast Reaction (mild or serious): 0.2-0.7 %
- Incidence of Serious Contrast Reactions: 0.01 -0.02 %

Who gets contrast reactions?

- Greatest risk is prior reaction (5x increase for reaction).
- Atopy (2-3x increased risk of a serious reaction)

What is the idea behind premedicating?

- The goal is to block histamine. This is typically done with a steroid + an antihistamine.

Routine Premedication		
Method 1:	Prednisone: 50 mg by mouth at 13 hours, 7 hours, and 1 hour before contrast	Diphenhydramine (Benadryl): 50 mg IV (or PO) 1 hour before contrast
Method 2:	Methylprednisolone: 32 mg by mouth 12 hours and 2 hours before contrast	Diphenhydramine (Benadryl): 50 mg IV (or PO) 1 hour before contrast
Emergent Premedication		
Method 1:	Methylprednisolone 40 mg or hydrocortisone sodium succinate 200 mg IV Q4 until contrast study	Diphenhydramine (Benadryl) 50 mg IV 1 hour prior to contrast injection;
Method 2: * <i>For Allergy to NSAIDS, or history of Asthma</i>	Dexamethasone sodium 7.5 mg or betamethasone 6.0 mg IV Q4 until contrast study.	Diphenhydramine (Benadryl) 50 mg IV 1 hour prior to contrast injection
Method 3: * <i>For People who can 't/shouldn't get steroids - infected, or at risk for bowel perforation</i>	Diphenhydramine 50 mg IV 1 hour prior to contrast injection.	

Contrast Reaction Management

Reaction	Treatment
Urticaria	Stop Injection
	Most cases don't need treatment
	HI Blockers: Diphenhydramine (Benadryl) PO/IM/IV 25mg-50mg
Severe Disseminated Urticaria	Epinephrine SC (1:1,000) 0.1 to 0.3 ml (=0.1 to 0.3 mg)
Facial or Laryngeal Edema	O2 6 to 10 liters/min (via mask).
	epinephrine SC or IM (1:1,000) 0.1 to 0.3 ml (=0.1 to 0.3 mg) or, especially if hypotension evident, epinephrine (1:10,000) slowly IV 1 to 3 ml (=0.1 to 0.3 mg). Can repeat epi up to maximum of lmg.
Bronchospasm	O2 6 to 10 liters/min (via mask).
	Beta Agonist: Albuterol
	If unresponsive to Albuterol - epinephrine SC or IM (1:1,000) 0.1 to 0.3 ml (=0.1 to 0.3 mg) or, especially if hypotension evident, epinephrine (1:10,000) slowly IV 1 to 3 ml (=0.1 to 0.3 mg). Can repeat up to maximum of lmg.
Hypotension with Tachycardia	Legs elevated 60 degrees or more (preferred) or Trendelenburg position.
	Give O2 6 to 10 liters/min (via mask).
	Rapid intravenous administration of large volumes of Ringer's lactate or nonnal saline.
	If unresponsive to fluids - epinephrine SC or IM (1:1,000) 0.1 to 0.3 ml (=0.1 to 0.3 mg) or, especially if hypotension evident, epinephrine (1:10,000) slowly IV 1 to 3 ml (=0.1 to 0.3 mg). Can repeat up to maximum of lmg.
Hypotension with Bradycardia (vaso-vagal reaction)	Secure airway: give O2 6 to 10 liters/min (via mask).
	Legs elevated 60 degrees or more (preferred) or Trendelenburg position
	Rapid intravenous administration of large volumes of Ringer's lactate or normal saline.
	If unresponsive: Give atropine 0.6 to 1 mg IV slowly . Repeat atropine up to a total dose of 0.04 mg/kg (2 to 3 mg)
Seizure or Convulsion	Secure airway: give O2 6 to 10 liters/min (via mask).
	Diazepam (Valium) 5 mg IV (or more, as appropriate) or midazolam (Versed) 0.5 to 1 mg IV.
	If you need extended coverage - Phenytoin (Dilantin) infusion 15-18 mg/kg at 50mg/min.
	Monitor for respiratory depression when giving Benzos

Why is the concentration for IV Epi different than SubQ?

The way to think about this is that you need a large volume of fluid for IV - because you are going to push it around. Alternatively, you wouldn't want a large volume of fluid under the skin. The dose is actually the same (0.1 - 0.3mg) it's just the dilution volume that is different.

SC or IM: 1:1,000, 0.1 to 0.3 ml (0.1 to 0.3 mg)

IV: 1:10,000 , 1 to 3 ml (0.1 to 0.3 mg)

Contrast Induced Nephropathy (CIN):

Defining CIN?

The stone age ("historic") criteria is an absolute increase in the serum creatinine from by 0.5 mg/dL. There are more modern definitions that require an absolute increase of up to 2.0 mg/dL, or a percentage of change in the baseline serum creatinine of 25 to 50 percent - depending on the study. The reality is CIN is way way way over diagnosed, and there are at least 2 large studies showing that most things that are called "CIN" are just normal variation in Cr that occur in patients who are sick in the hospital.

How often does CIN occur? Real answer is almost never. If asked on the CORE, I would say something like less than 10% even with moderate kidney disease.

When does CIN occur? The typical course is a spike in Cr within 24 hours of getting contrast, peaking around day 4, then returning to baseline at day 10.

When should you check a Cr?

- * Age >60;
- * History of renal disease (dialysis, renal transplant, renal cancer)
- * Solitary Kidney
- * Bad Hypertension (that requires medication)
- * Diabetes Mellitus
- * Metformin

What is the deal with metformin? The trick is metformin is NOT a risk for developing CIN. However, if you do develop renal failure while on metformin you could get a lactic acidosis.

What do you do if you are on metformin? The typical recommendation is to stop the drug at the time of the study and hold in for 48 hours. If they didn't develop CIN - they can restart after 48 hours.

How Does One Prevent the Plague of CIN? There are several techniques all of which are bogus. However, for the purpose of the CORE intravenous hydration, preferably with isotonic fluids such as 0.9% saline or Lactated Ringer's at 100 ml/hr for 6-12 hour before contrast administration and 4-12 hours after contrast administration is the best. The reason it "works" is that it artificially lowers the Cr on the labs.

Contrast Extravasation:

What is the typical outcome? Most of the time there is no significant sequelae.

Is the rate of injection a risk factor? No... but, it can make the severity worse.

What are the "severe" complications?

Compartment Syndrome - This is the most common, and related to mechanical compression. The primary risk factor is the volume of contrast extravasation, into a small compartment (hand, foot, or ankle).

Skin Ulceration / Necrosis -

What is the treatment? There is no consensus - so you shouldn't get ask. If you do get asked, I guess I'd say elevate the affected extremity above the heart.

Gadolinium Contrast agents:

Gd is easier than iodine contrast agents, with regard to necessary trivia. Just remember these things:

Don't give Gd to patients with a GFR <30 or with acute kidney injury.

Check a GFR (within 6 weeks is adequate) with patients with renal disease, transplant, over the age of 60, diabetes, or a history of hypertension.

Section 3: Radiation Safety - Buzzwords, Marketing, and Trivia

Buzzwords - All of which mean the same thing:

“Image Gently” Pediatrics

“Image Wisely” Adult equivalent

“Step Lightly” Reduce radiation in pediatric IR. “Step lightly on the fluoro”

“ALARA” As low as reasonably achievable. Basically try and minimize radiation dose.

Biologic Effects of Ionizing Radiation (BEIR) Committee - Also known as BEIR 7 Report

What were the sources of data used? Atomic Bomb Survivor data, medical radiation studies, occupational radiation studies, and environmental radiation studies. **The primary source was the bomb data.**

What was the primary task of this committee? To develop the best possible risk estimate for human exposure to low-dose (<100 mSv), low-LET ionizing radiation.

What did the BEIR 7 Report conclude? That there is a thing called a linear, no-threshold dose response between radiation and the development of cancer in humans.

More specifically, what does BEIR 7 accuse medical imaging of? Giving everyone cancer. BEIR 7 predicts that 1 in 100 will get a solid cancer or leukemia from a dose of 100mSv above background.

Is BEIR 7 legit? No... it's probably a load of monkey poop. If you look at how they get their data, they use a risk model that is “x” times excess relative risk + (1-“x”) times the excess absolute risk. In this equation, “x” is determined by committee! In other words... they just made it up. Basically, all estimates are based on multiple models and assumption with confidence intervals that are subjective and partly based on opinion. Cancer estimates from BEIR 7 are not proven facts.... Having said all that, the test will probably want you to treat them as such. Who knows... **just say linear no-threshold based on bomb data.**

Section 4: “The Trainee Caused a Complication”

Any time I enter a patient care area, the first thing I do is look for the person I’m going to blame if something goes wrong- usually a tech or nurse. If I were working in academics, I would choose a “Trainee.”

What is a "Trainee ? " A Trainee is the new word for resident. It was developed by bureaucrats to help maintain the hierarchy of academic medicine, with the eventual goal of providing no salary or benefits (why would you pay the trainee?).

When can a “Trainee ” be referred to as a Doctor? There are only two scenarios. (1) If there is a menial task that requires a physician signature (writing scripts, doing H&Ps, consenting patients, etc....) or (2) there has been a complication and someone needs to be blamed.

National Patient Safety Goals:

You need two identifiers when providing patient care / doing a procedure.

Labeling Medications: Current Medication Labeling Rules *{this isn’t Nam there are rules}*:

- * Meds on and off the sterile field are labeled
- * Meds are labeled even if they are the only ones on the field
- * Drug Name and Strength (concentration) should be on the label
- * You can NOT pre-label empty syringes
- * Medications used throughout a procedure (pre-solution, normal saline for rinsing stuff, etc...) must be labeled - or the receiving container must be labeled.
- * Exception to labeling rule is a scenario when you draw something up from the original container and immediately administer it.

Communicating Critical Results:

Per the National Patient Safety Goals (02.03.01) - Written procedures for dealing with critical results should be developed by the radiology group, which include to who from who, and the acceptable length of time between the read and the call.

What is this “Error” You Speak Of?

Types of Error: As stated above, most occur as a “system” and not individual. For the purpose of multiple choice, you can group these into two categories.

- * Active Errors - Errors at the point of contact.
- Latent Errors - Errors which occur because of organizational failure (bad staffing, unsafe environment). An “accident waiting to happen.”

Vocabulary of Error

Adverse Error: Any kind of iatrogenic injury. This doesn’t necessarily mean an error occurred, it could be a side effect or therapy or known complication of the procedure. Examples include, drug allergy or post op infection.

Blunt End - The part of the health care system that is NOT in direct contact with patients.

Examples: Various bureaucrats, bean counters, chairmen. The people who set policy.

Sharp End - The part of the health care system that is in direct contact with stinky patients

Examples: Various Trainees, nurses etc...

Mistake: An error that occurs because of insufficient knowledge (you picked the wrong antibiotic, or chose the wrong test).

Slip: An error that occurs because of a lapse in concentration.

Near Miss (*Close Call*) - An event that could have caused an injury to the patient, and only didn’t because of pure dumb luck.

Sentinel Event: An event that causes death , serious harm, or almost causes death/harm.

Classic Radiology Sentinel Event = whole body radiation dose of 15 Gy.

Why Errors Occur?

“*To Err is Human*” - This was a project started by the National Academy of Sciences’ Institute of Medicine. The paper said most errors were system errors rather than individual errors. The result was the spending of 50 million dollars by congress to create a list of “never events” which require mandatory reporting.

Institute of Medicine *patients die from system errors > list of never events.*

Behaviors that lead to error:

- * **Human Error** - This is an unintentional and unpredictable behavior that causes an unwanted outcome. *You reached for the glass of milk, and spilled it.* These types of errors occur because of weakness in the system (the cup needed a lid, or the bottom of the cup should be been wider).
- * **At Risk Behavior** - This is an unsafe habit, which the person thinks is a justified risk. *“This is just the way we do things here.”* I always reach for my milk while I’m watching tv. It’s ok, I won’t spill it... I’ve been doing this for years.
- * **Reckless Behavior** - This time the worker knows there is risk, and understands it is a real risk. They can’t really defend why they did something. *I just thought it would be funny to balance the cup of milk on my nose.*

A Just Culture: This is a concept of accountability that distinguishes human errors, at risk behaviors, and reckless behaviors and addresses them accordingly.

Just Culture	
Human Error	Console the worker. Then try and fix the system.
At Risk	Don’t Punish. Instead, fix the system based reason for behavior. Work to decrease the acceptance of such behavior from the staff.
Reckless	Punish!!! It’s not revenge, it’s punishment... according to department policy (placing the offender in the stockade).

What is this “Safety” You Speak Of?

Culture of Safety

The general concept is that the administration recognizes that what you do is high risk (needle sticks for HIV/ Hep C patients, constant threat of litigation, the ED being stupid, harassment from the trauma team). Because the administration agrees that things are high risk, it wants people to report misses or near misses so that they can be corrected. The culture of free reporting means that there is a blame-free culture. You won't get in trouble as long as you tell the truth. In the same vein as “all evaluations are anonymous.”

Safety Champion: The Joint Commission describes this idea of a “safety champion.” This is someone selected from each unit or department that receives special training to help advocate for safety and work environment awareness. They will help educate other members of the department and report them for “unsafe behaviors.”

Environmental Safety Tours: The Joint Commission requires two environmental tours in the patient care area and one tour in non-patient care areas per year. The “safety champion” can be the one to do this.

Testable Trivia:

- The title of safety champion should rotate among staff
- The safety officer should train the safety champion
- The safety officer can do the environmental tours (2 patient care, 1 non-patient care).

Stuff Nurses Do:

Medication Reconciliation - This is a task nurses who work on medical surgical floors will do to help reduce medication errors. Grandma will come in with a list of about 50 medications, many of which she isn't taking, many of which are duplicates with both generic and brand names listed separately (with different doses), etc....

The process of fixing this can be terrible, because if you ask Grandma what she takes she will probably tell you “I take a blue pill in the morning, and that orange one at night....”.

Prior to discharge it's good to go through the list and make sure it makes sense, including the new medications she'll be on after discharge. Luckily, you are not a nurse or a medicine doctor, so you will never have to do this.

Buzzwords For Diagnosing and Preventing Errors

Human Factor Engineering - The idea that humans make mistakes, so you should build the system to minimize the risk of error.

Usability Testing - This is the idea of *testing systems in a real world condition*. The classic example is looking at the computerized provider order entry (CPOE) system, to see if it makes work harder (reducing bedside patient time) or actually creates more errors.

Workaround - This term describes workers not following the rules. They “work around” the rules, to get things done quicker.

Forcing Function - This term describes not allowing errors to occur to start with. For example, you don’t stock lethal injection materials (concentrated potassium) in the code box.

Standardization - Making everything the same way, and **using lots and lots of check lists** is supposed to decrease the likelihood a trainee will make an error.

Resiliency Efforts -The general philosophy here is that trainees make errors and there is just no way to stop that. Instead, energy is focused on detecting errors early and stopping them before they progress to something worse.

Failure Mode and Effects Analysis (FEMA) - Prospectively identify error risk within a particular process

Root Cause Analysis - Retrospective; structured method used to analyze serious adverse events.

This vs That	
FEMA	Prospective
Root Cause Analysis	Retrospective

Section 5: “Getting Sued”

Since you made the terrible mistake of being a doctor, your punishment is to spend your career worrying about getting sued and eventually getting sued. This can only be prevented by (1) working at a VA, or (2) quitting medicine and going to law school (so you can do the suing).

What gets you sued?

- * Errors in diagnosis are the most common cause of malpractice suits against Radiologists
- * Imaging findings related to breast cancer is the most common organ related subject of malpractice suits against Radiologists
- * Communication Errors (with referring M.D., or failure to recommend another test) is actually a much less frequent cause of litigation - when compared to interpretive error.

Whang, Jeremy S., et al. "The causes of medical malpractice suits against radiologists in the United States." Radiology 266.2 (2013): 548-554.

What needs to be proved?

All 5 Elements must be proved for a successful medical malpractice case:

1. A duty was owed: You owe that duty once you read that case, or do that procedure.
2. A duty was breached: You didn't follow the standard of care.
3. The breach caused an injury: Your failure to follow the standard of care is what hurt the patient.
4. Deviation from the accepted standard: You didn't follow the standard of care in your specialty (this is similar to #2).
5. Damage: The patient must have gotten hurt (physical or emotional). If they didn't get hurt, it doesn't matter if you were negligent or not.

Basically, it was your case, you missed the finding or didn't communicate a critical finding (or other violation of the standard of care), and the patient got hurt. If they didn't get hurt, then you are ok. If they got hurt but you followed the standard of care you are ok.

Section 6: “Getting Paid”

The only reason to go to medical school and endure residency / fellowship is the chance to make enough money to buy a Porsche and/or Boat. This is still not guaranteed, as hospital administrators make more and more and doctors make less and less every year.

Step 1: Getting Credentialed

Testable Trivia resolves around two process:

- (1) ***Focused Professional Practice Evaluation (FPPE)*** - This is basically current medical staff checking your work (proctoring) you for the first few studies you read or a committee evaluating if you can do a new procedure or not.
- (2) ***Ongoing Professional Practice Evaluations (OPPE)*** - The a continued review of your performance - typically q6 months, that is used for reappointment. Your department determines the scope of the review.

Step 2: Getting A Paycheck

Testable Trivia Regarding Payment:

Two Lists of Diseases/Procedures Made by Two Different Bodies for Billing:

1. Current Procedural Terminology Codes (CPT) - The **AMA editorial panel** is actually in charge of these. They create a “uniform” description of the procedure.
2. International Classification of Diseases (ICD) 9 - This is a list of diseases made by the **World Health Organization (WHO)**. The “9th” version of this list is the most popular one.

Two Ways the Government Punishes you for Going to Medical School:

1. Physician Quality Reporting System (PQRS) - Medicare uses carrots (stick to come later) to force you to report “quality clinical data” on certain conditions.
2. Meaningful Use - Forcing you to have an electronic medical record, and then prove (by random button clicking) that you are using it.

Resource Based Relative Value System (RBRVS) - This is a system based on some Harvard study , that said doctors should get paid based on the time it took to do something and how difficult it was to do it. RBRVSs are adjusted based on geography with a “Geographic Practice Cost Indicator”, so that you make more in LA than you do in rural South Dakota or New Mexico

Omnibus Budget Reconciliation Act of 1989 (OBRA 89) - This is the name of the Medicare payment reform law.

Participating vs Non-Participating Physicians

Participating - If you agree to take what medicare gives you and not try and get the rest of the money owed to you by the patient.

- Non- Participating - You refuse to sign the agreement, and can attempt to collect the rest of the money from the patient. The government tries to discourage this by only paying you 95% of what is paid to the participating physicians.

Sustainable Growth Rate System (SGR) - This was put in place in 1997 to control the growth of Medicare. It’s based on domestic product per capita. This is totally flawed, and would result in you making no money at all and being homeless eating out of the dumpster behind McDonalds. The only thing that keeps you from enjoying two day old Big Macs is a “patch fix” congress does yearly (at the stroke of midnight). The AMA wants a repeal of the SGR, but currently the AMA lacks enough funds to “convince” members of congress to act on its behalf.

Section 7: Radom Trivia

“Off Label”

- Using an approved drug / device for something other than its original intended use is called “off label” use.
- The FDA is in charge of monitoring the safety of medical devices and drugs
- The “*Practice of Medicine* ” doctrine allows doctors to use things off label.
- For regular clinical work, off label use does NOT have to be disclosed while obtaining informed consent
- For research studies, off label use MUST be disclosed while obtaining consent
- If you are using a medication / device off label, you can’t market the product as your own special product.

ACR Appropriateness Criteria

In an attempt to get promoted, various academic Radiologists got together in 1993 and formed committees to decide what tests are appropriate. The idea was to help ordering providers get the right test.

Testable trivia:

- Appropriateness Rating
 - Rating 1-3 = Usually NOT appropriate
 - Rating 4-6 = Might be appropriate
 - Rating 7-9 = Usually appropriate
- There is also a 1 -6 Relative Radiation Score for each test.

Work Related Problems for Radiologists

Stress Over Proving You are a “Real Doctor”

- Improved by - getting control of your ego. Remember your alternative choice in medicine was to dedicate your life to poop, pus, and note writing.

EyeStrain Asthenopia”

- According to an article from AJR 2005, written by a Canadian Academic Center - you can reduce eye strain by taking short breaks, reading less CTs, working less than 7 hours a day, and eliminating screen flicker. When I read this paper I had to laugh. Are you seriously that weak? Work hard! Make Money! Retire! In that order. Stop crying about your eyes hurting.

Carpal Tunnel Syndrome:

- Too much mousing

Back Pain

- Most common in IR - from the lead

CPR:

They have changed the CPR rules.... Again. Don't worry I'm sure the changes are evidence based.

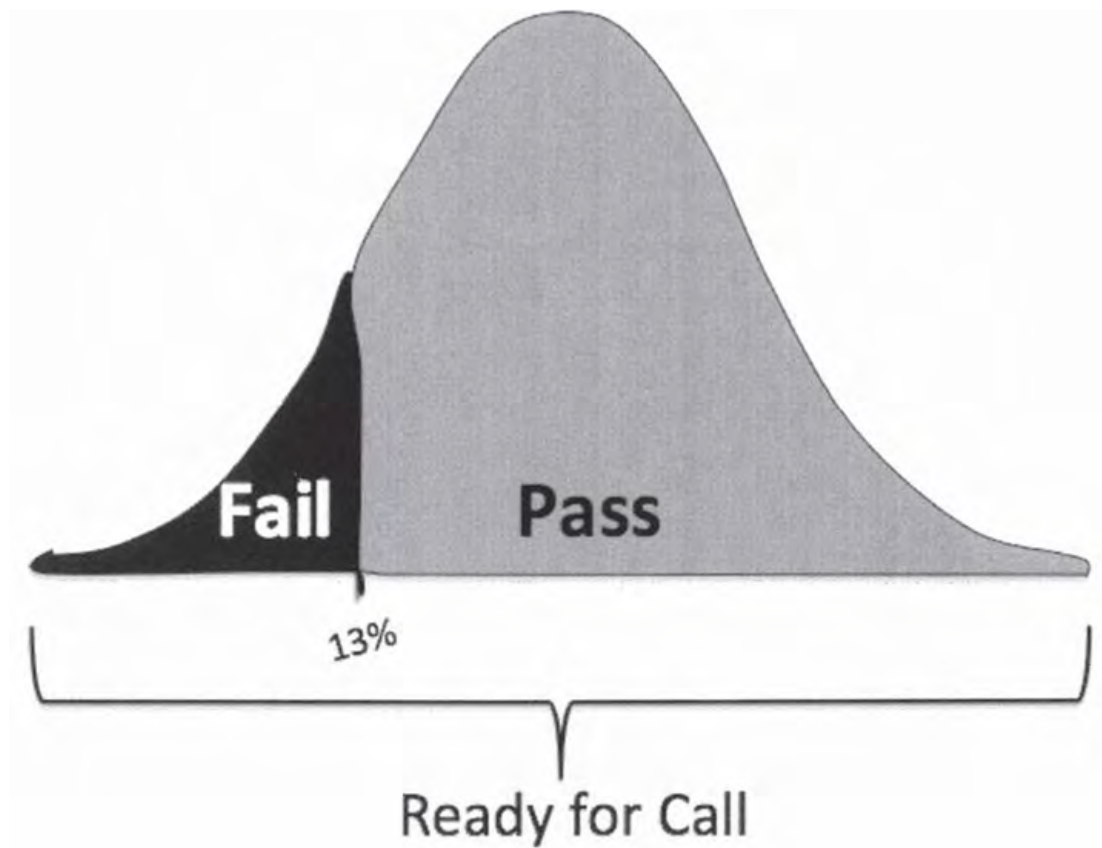
Testable Trivia:

- * *Current Sequence:* (1) Chest Compressions, (2) Airways, (3) Breathing. The American Heart Association believes it's critical to survival to begin circulating un- oxygenated blood first.
- * Compression rate is 100/min, at 2 inches depth (to the beat of the disco song staying alive).
- * Ratio is always 30:2 with one exception; the two person pediatric which is 15:2

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Biostatistics

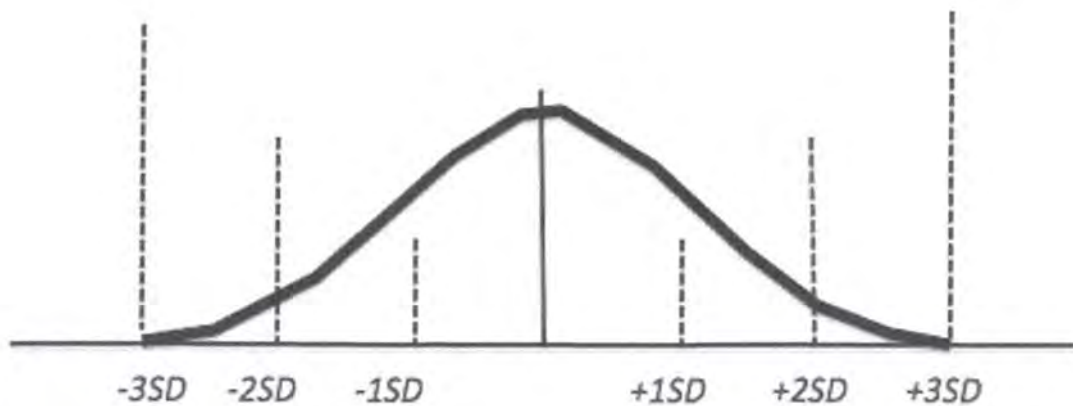
Prometheus Lionhart, M.D.



There is just no escape from this crap. It has been on every major certifying / board exam you've taken, and will be on every one to follow.

The Normal Distribution:

This will look familiar as it is how every “good test” will turn out. Naturally occurring phenomena will distribute as a bell-shaped “normal” or “Gaussian Distribution.”



Data can be “skewed” to one side or the other.

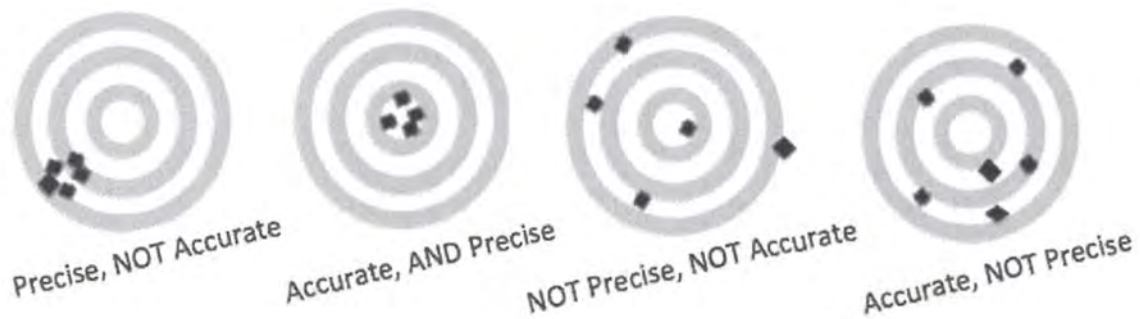


The “negative” or “positive” skewing is described by which side the tail is on. The relationship to the mean is the most likely testable trivia. A negative skew has the mean after the tail. A positive skew has the mean before the tail.

Precision and Accuracy:

Precision - This is the immunity to variation. “The dart hits the same spot every time.” A wider confidence interval = a less precise study.

Accuracy (unbiased) - This is the immunity to systematic error or bias. “The dart hits the center.”



What is this “Statistical Significance” ?

You often hear people say “the study was significant, with a p value < 0.05 .” What the hell does that mean anyway? Basically, they are saying that the result is unlikely to have occurred by chance. You can flip a coin that lands on heads 50 times in a row, it’s just not very likely to occur by chance. If you say the p value is < 0.05 , then you are saying the likelihood it occurred by chance is less than 5%. In other words, the investigator is 95% sure the result did NOT occur by chance.

Correlation and Causality:

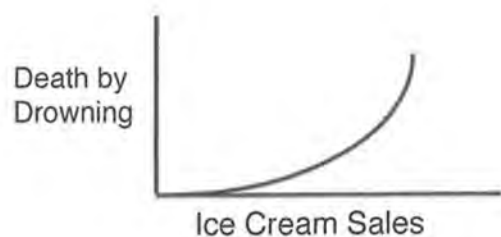
Just because two things seem to rise together (correlation), doesn’t mean one is causing the other. The example I like to use is ice cream sales and death by drowning. You will find that the more ice cream is sold, the more people die in swimming pools and the ocean. Why is ice cream so deadly? Should we ban it? The people who believe the MMR vaccine causes autism probably think we should, but they don’t understand the difference between correlation and causality.

When do people buy ice cream ? — The Summer

When do people go swimming? - The Summer

Warm weather is actually the reason these things go up together, they are not actually causing each other.

Statistical Epidemiology



Incidence: The number of NEW cases occurring in a particular time period.

$$\text{Incidence Rate} = \frac{\text{Number of NEW cases of disease}}{\text{Total Number of People at Risk}} \text{ Per unit time}$$

Prevalence: The number of cases of a certain disease in a particular moment in time.

$$\text{Prevalence Rate} = \frac{\text{Number of cases of disease}}{\text{Total Number of People at Risk}} \text{ At a particular time}$$

Sensitivity & Specificity

Both sensitivity and specificity measure validity (the ability to detect people with or without disease).

True Positive (TP) = Test shows cancer, Patient has cancer

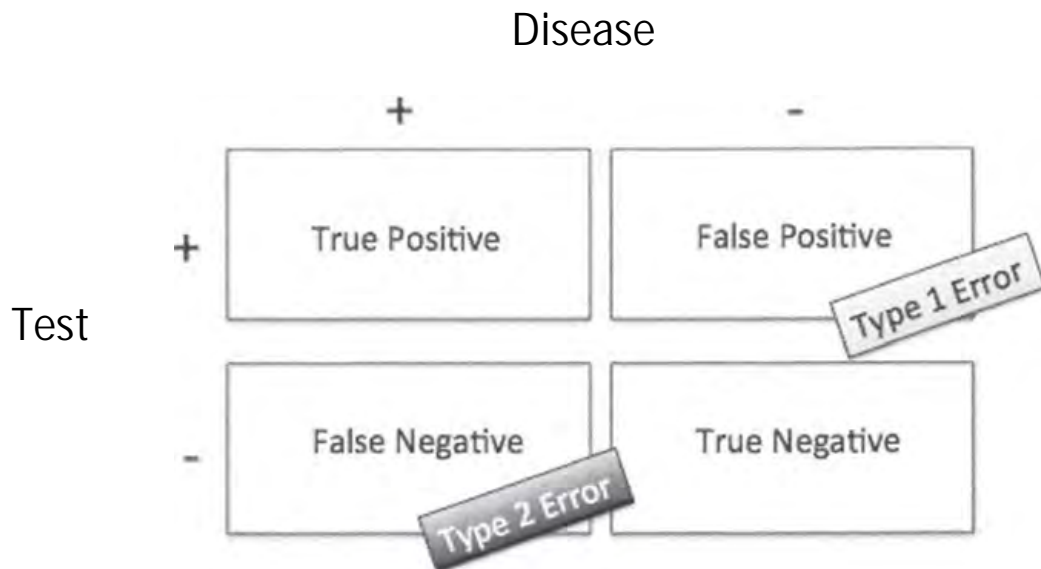
False Positive (FP) = Test shows cancer, Patient does NOT have cancer.

** Same as a Type I error.*

True Negative (TN) = Test shows NO cancer, Patient does NOT have cancer.

False Negative (FN) = Test shows NO cancer, Patient has cancer.

** Same as a Type II error.*



I want to discourage you from using the A, B, C, D method for doing statical problems. This can get you in hot water if the question writer flips the axis of the square (which they love to do). It's just better to understand what you are actually measuring.

Sensitivity = This is the *ability of the test to detect true disease*. In other words, the amount of true disease detected with the test / total people with the disease.

$\text{True Positive} / \text{True Positive} + \text{False Negatives}$.

Specificity = The is the *ability of the test to detect people free of disease*. In other words, the amount of disease free people called negative by the test / total number of disease free people.

$\text{True Negative} / \text{True Negative} + \text{False Positives}$

A very specific test rules in disease. (SPIN ; SPecificity rules IN)

— Type 1 Error : The fire alarm goes off, but there is no fire. - A false positive.

— Type 2 Error: A fire burns, but the alarm does not go off. - A false negative.
Obviously, this is the worst kind of error.

Accuracy = Think about this as how often the test was right:

$$\text{True Positive} + \text{True Negative} / \text{TP} + \text{FP} + \text{TN} + \text{FN}$$

Positive Predictive Value: This is the likelihood that a person with a positive test actually has the disease.

$$\text{True Positive} / \text{True Positive} + \text{False Positives}$$

Negative Predictive Value: This is the likelihood that a person with a negative test actually is disease free.

$$\text{True Negative} / \text{True Negative} + \text{False Negative}$$

Sensitivity and Specificity depend only on the characteristics of the test. Predictive value depends on the prevalence. This is a concept that multiple choice writers love!

The higher the prevalence of a disease the higher the PPV and the Lower the NPV.
The lower the prevalence of a disease the lower the PPV and the higher the NPV.

For Example, Let's say there is a very rare disease called "The Fever." The symptoms of this include being paid very large amounts of money to screw over the next generation of your colleagues.

A very specific test might still have a low PPV because it's going to have a lot of false positives. In other words if the prevalence is 1:300,000,000 and your false positive rate is 1:1,000,000 you are still going to have 1 true positive, and 299 false positives.

Validity - Is the test doing what it claims it does? An x-ray is a pretty valid test for looking for a fracture (it has the ability to show who has a fracture and who does not). Important qualities of a valid test are that it is - highly specific and highly sensitive.

Validity is similar to accuracy.

Power - Type II errors are bad (there is a fire, but the alarm doesn't go off). The ability to prevent this from happening is called "power." By convention a study needs a power of 0.8 (80% chance of the fire alarm going off for a fire). The most important thing to remember is that, the larger the sample size the greater the power. Larger protests have more power.

Risk:

Absolute Risk: The more common a disease is, the higher the risk of catching it. Most people will say that the incidence of a disease is the most important risk factor. That is why epidemiologists defined “absolute risk” as essentially the same thing as disease incidence.

The incidence of disease is 1:100, then the absolute risk is 1:100.

Relative Risk: Ever wondered how many times the guy with multi-drug resistant TB can cough on you on your plane ride to Chicago (*or Tucson*) to take the CORE exam before your PPD converts? This is the best way to think about relative risk - exposure to risk factors increases the risk of getting the disease.

RR = Incidence of disease among persons exposed to risk factor / Incidence of disease among people who did NOT get exposed to risk factor.

Absolute Risk Reduction: The general way to think about this is the difference between the people who did not get the drug (control group event rate) and the people who did get the drug (experimental group event rate). This is the inverse of number needed to treat.

Number Needed to Treat: How many drug addicts do you need to put in rehab before you actually get one clean? The math requires knowing the absolute risk reduction.

$$NNT = 100/\text{Absolute Risk Reduction}.$$

Attributable Risk: How much lung cancer is attributable to smoking?

$$AR = (\text{Incidence of disease in exposed}) - (\text{Incidence of disease in those NOT exposed.})$$

Odds Ratio: The absolute risk calculations require a prospective study. Odds ratio can be done using a retrospective study.

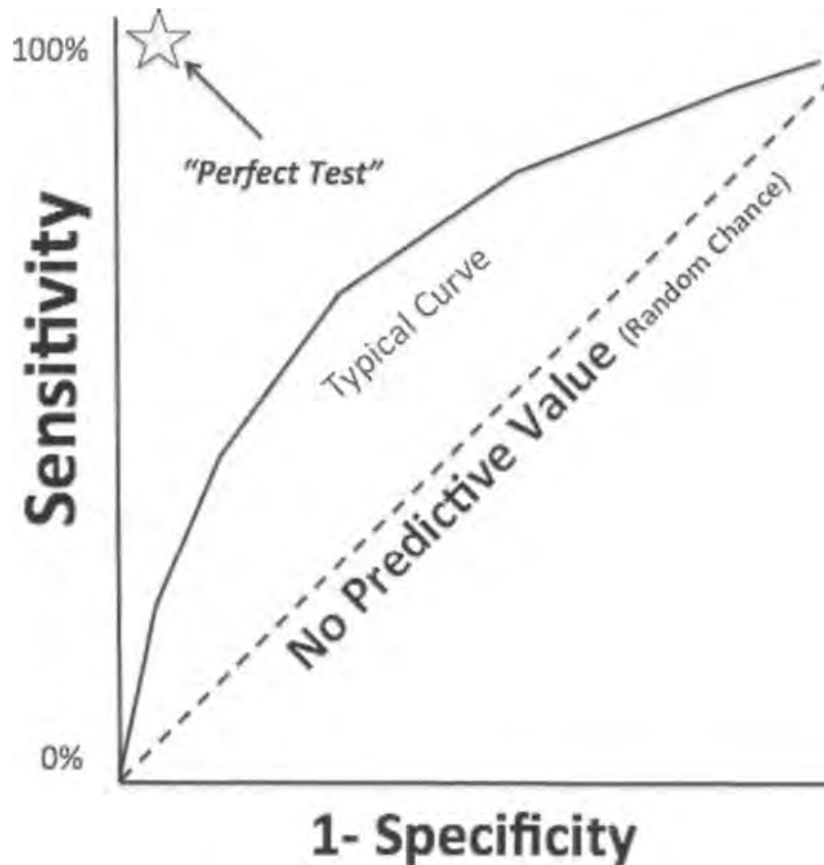
OR = Odds that a case was exposed to the risk factor

Odds that a control was exposed to the risk factor.

$$\text{OR} = \frac{(\text{Cases exposed to TB cougher on plane}) \times (\text{Cases of people not exposed})}{(\text{Controls exposed to the TB cougher on the plane}) \times (\text{Cases not exposed})}$$

ROC Curves:

People who write standardized tests have a strange obsession with ROC curves.



Questions You Can Ask About ROC Curves:

- (1) What are the axes of the graph ? Sensitivity, and 1-Specificity
- (2) What kind of a line would have no predictive value ? The straight one as above
- (3) What kind of a line would occur from random chance? Same question, just different wording - still the straight one as above.
- (4) Where is the "Ideal" or "Perfect" or "Gold Standard" test? Top left corner.
- (5) When the accuracy improves how does the curve change? Shifts towards the upper left corner.